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PER

SURGERY

A Monthly Journal Devoted to the Art and Science of Surgery

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Original Communications

Etiology of Acute Pancreatitis. Rolf Lium, M.D., Portsmouth, N. H., and Stephen Maddock, M.D., Boston, Mass. ---- 593

A Combined Abdominothoracic Incision Particularly Adapted for Use in Total Gastrectomy and Esophagogastrectomy. Arnold J. Kremen, M.D., Minneapolis, Minn. ---- 605

Gastric Resection: The Schoemaker-Billroth I Operation. John F. Higginson, M.D., and O. Theron Clagett, M.D., Rochester, Minn. ---- 613

A Suction and Feeding Tube for the Postoperative Care of Gastric Resections. Lawrence Singmaster, M.D., Philadelphia, Pa. ---- 621

Diaphragmatic Hernia Following Subdiaphragmatic Vagotomy. John M. Beal, M.D., New York, N. Y. ---- 625

The Role of Pleural Exudation in Infection Following Pneumonectomy. John T. Small, M.D., and George M. Higgins, Ph.D., Rochester, Minn. ---- 628

The Effect of Streptomycin, Local and Systemic, on Contaminated, Sutured Wounds. Charles K. Kirby, M.D., James A. Dull, M.D., Harold E. Fulton, M.D., Edward B. Price, M.D., and Harold A. Zintel, M.D., D.Sc. (Med.), Philadelphia, Pa. ---- 647

A Method for Puncturing Blood Vessels, Located and Exposed by Incision. Heinrich Lamm, M.D., La Feria, Texas ---- 653

(Contents continued on inside back cover)

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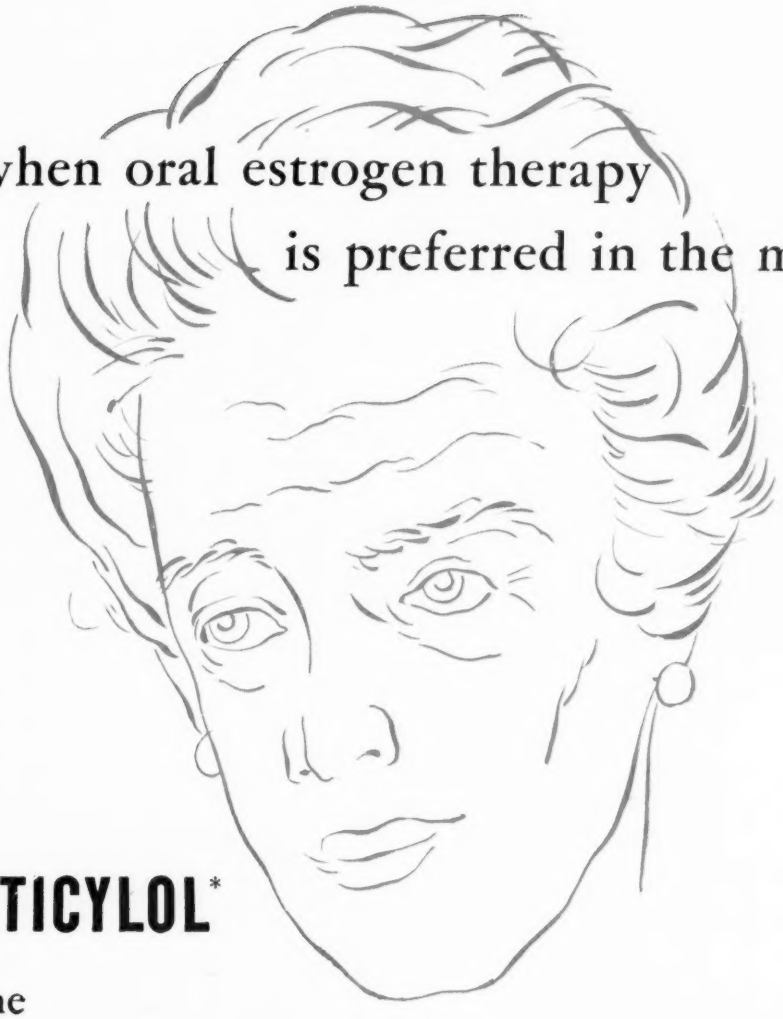

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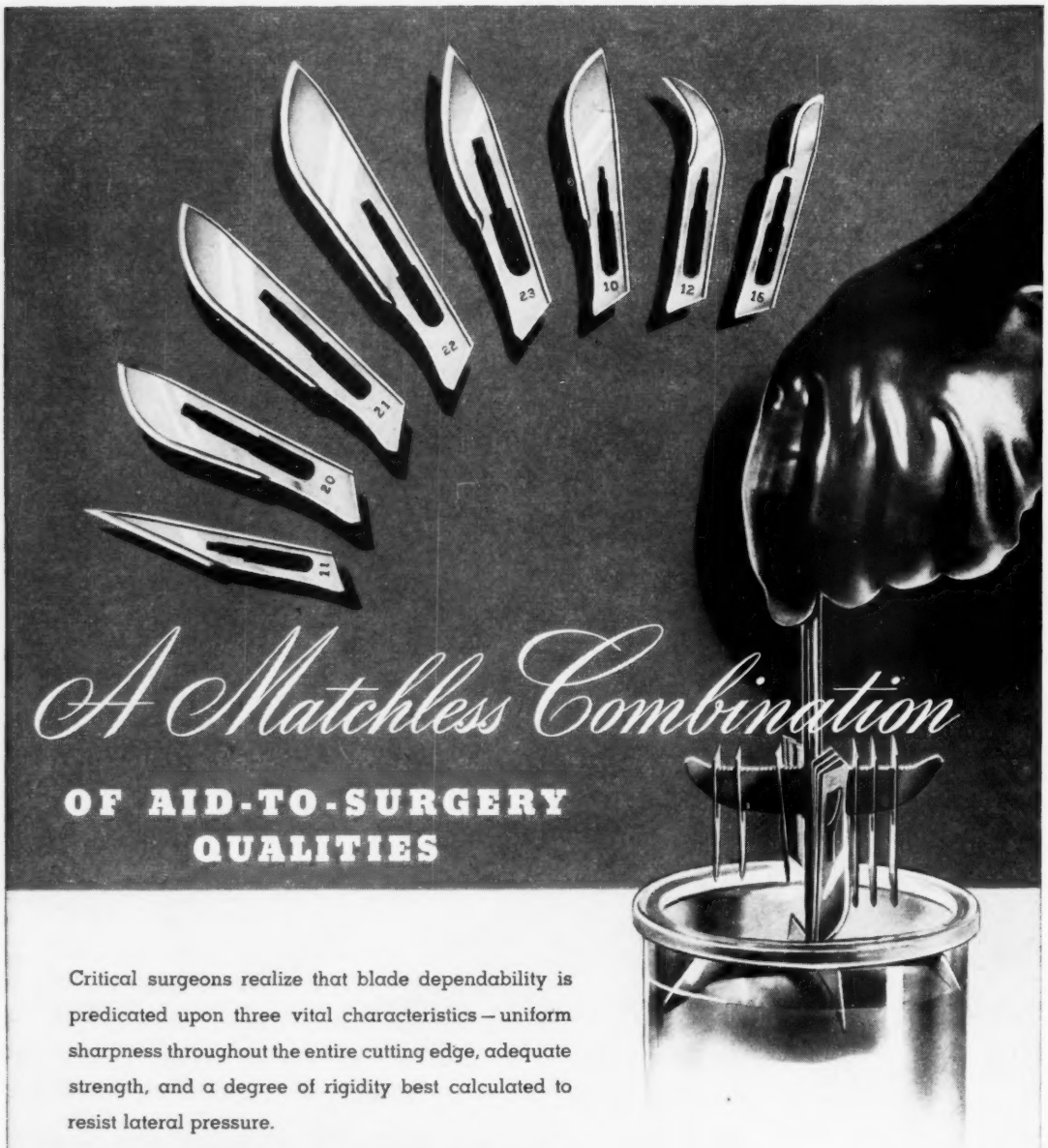

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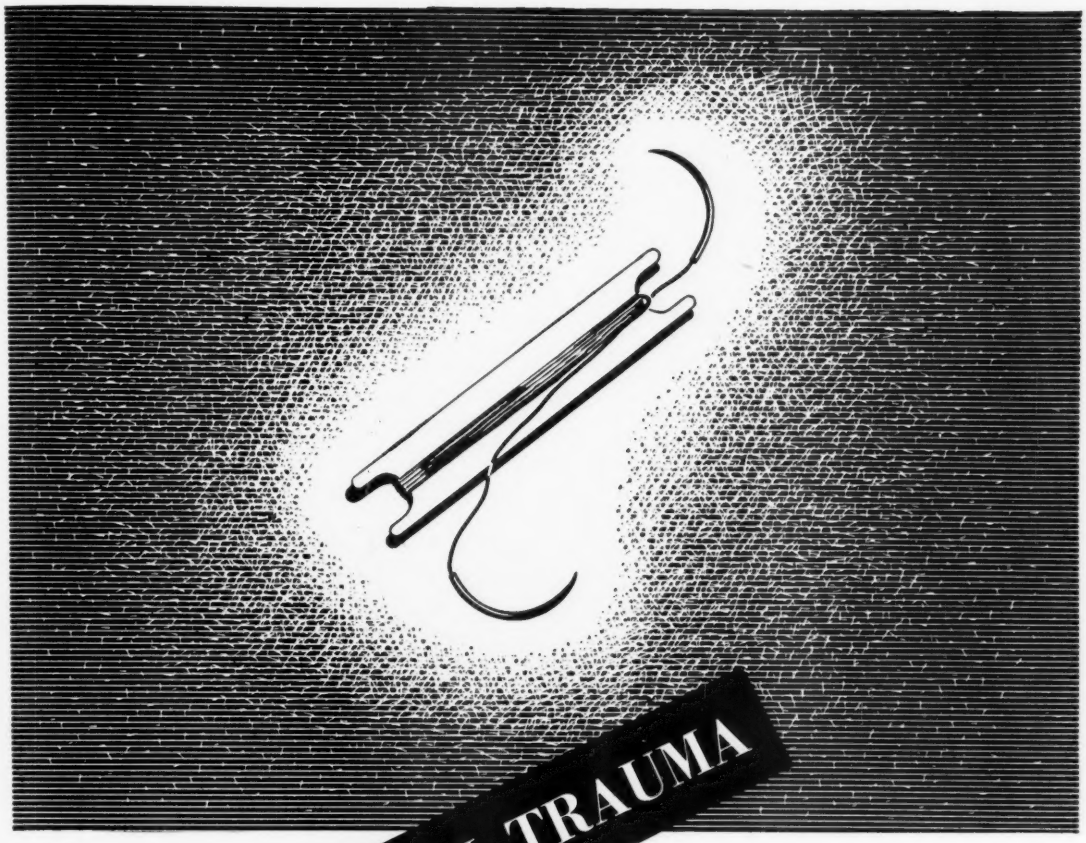

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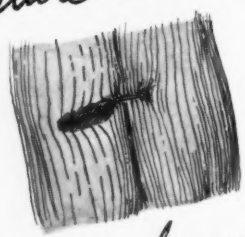


2. Knots become untied

Result of failure to tie 3 square knots

3. Knot becomes untied at end of continuous suture

Cannot occur with interrupted sutures

If one knot becomes untied, rest remain tied and carry on intended function

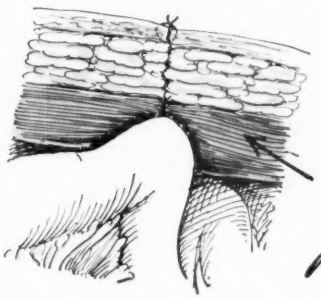
SUTUREGRAM

NUMBER NINE (CONTINUED)

Types of Wound Dehiscence

a) Separation of skin edges

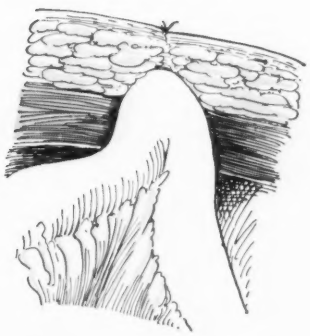
b) Post-operative hernia



Anterior sheath remains sutured and heals

Posterior sheath and peritoneum separate

c) Evisceration
All layers separate to the skin.



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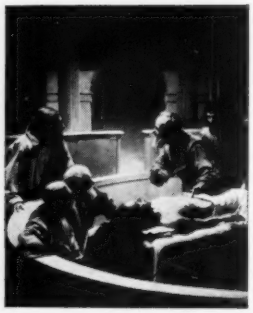

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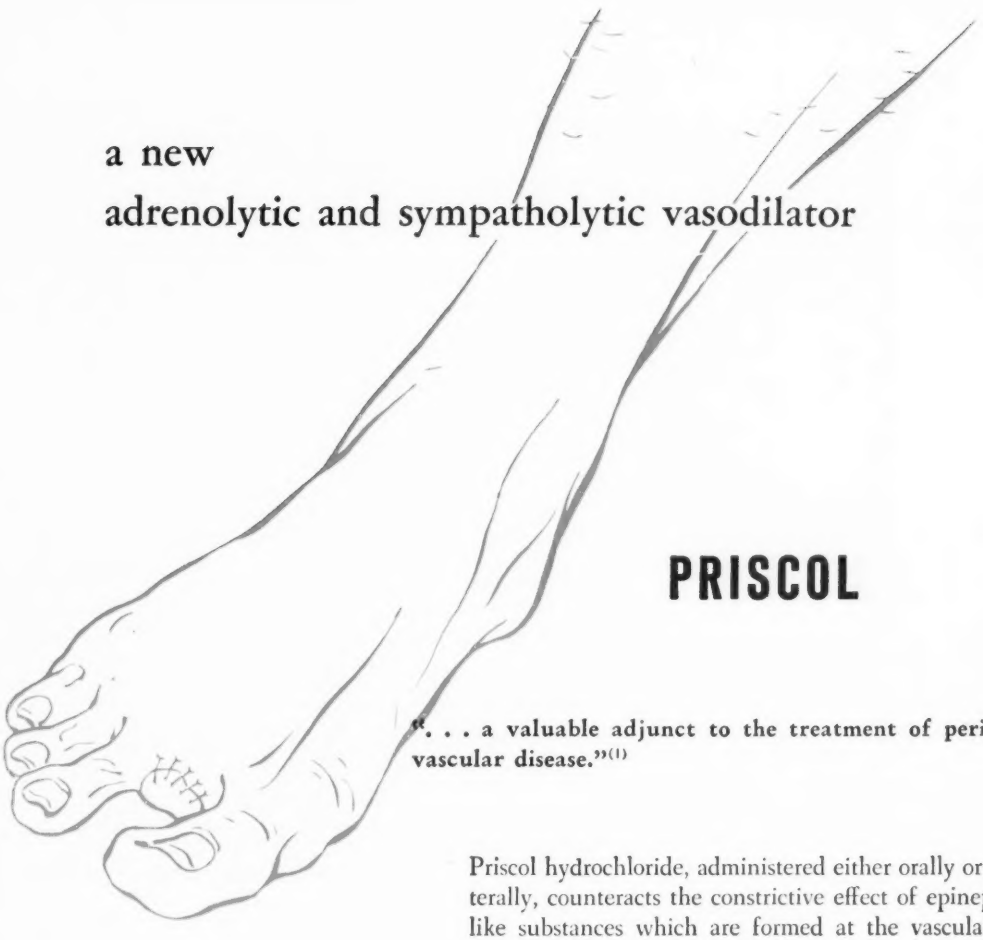

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1. Grimson, K. S., Marzoni, F. A., Reardon, M. J., and Hendrix, J. P.: *Surg.*, 23:728, 1948.

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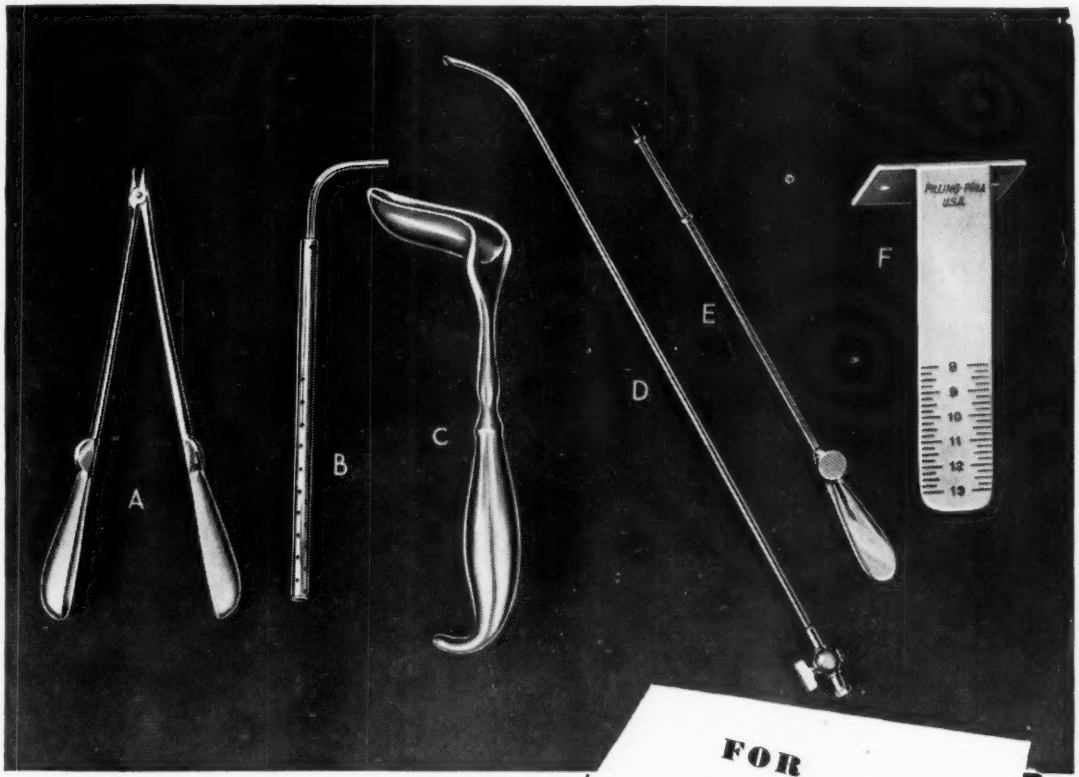

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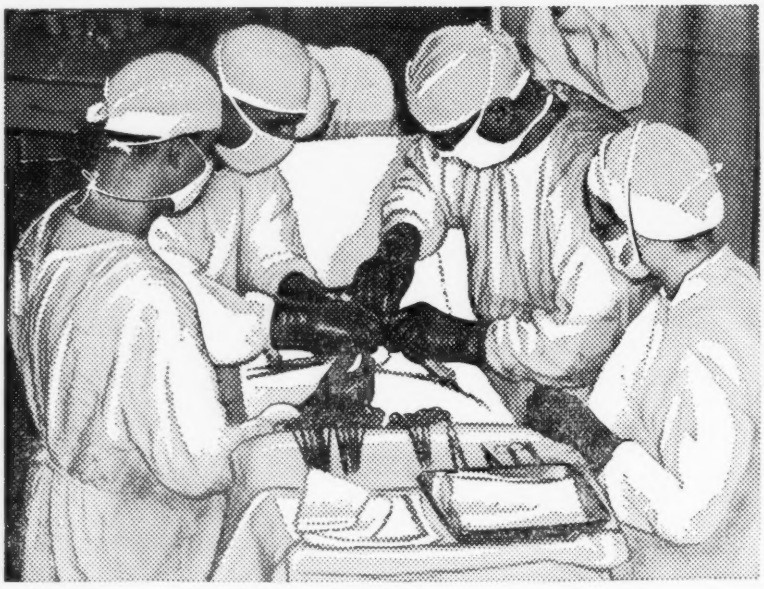

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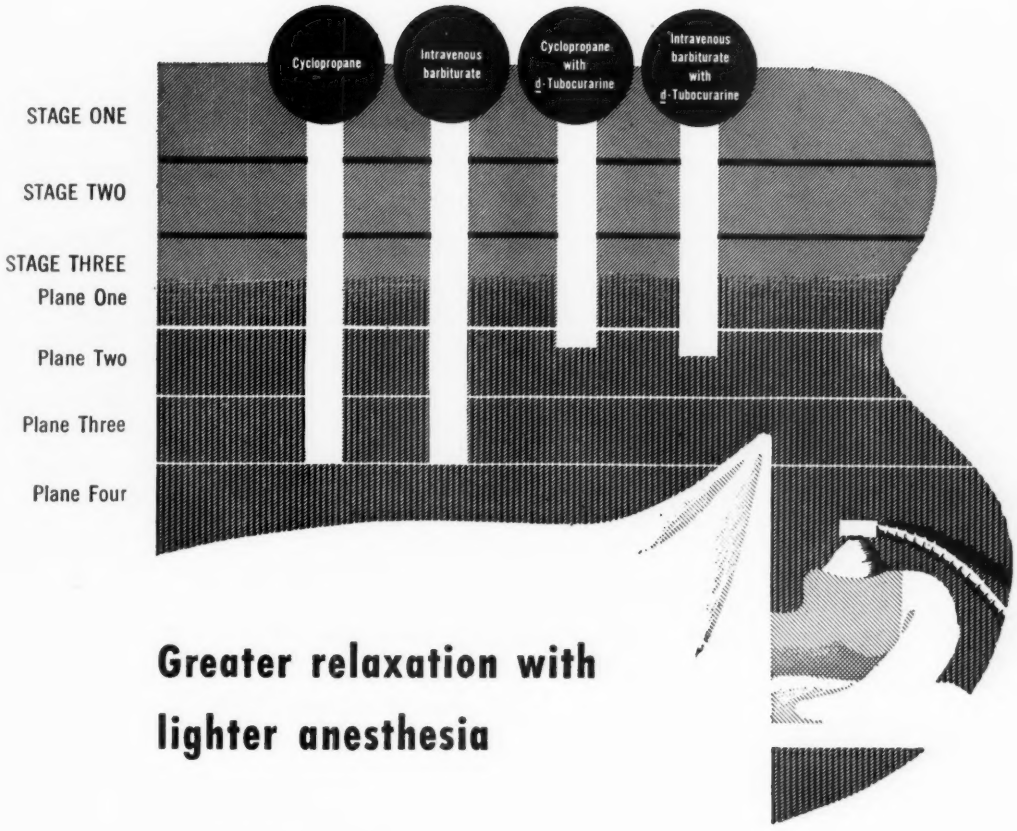

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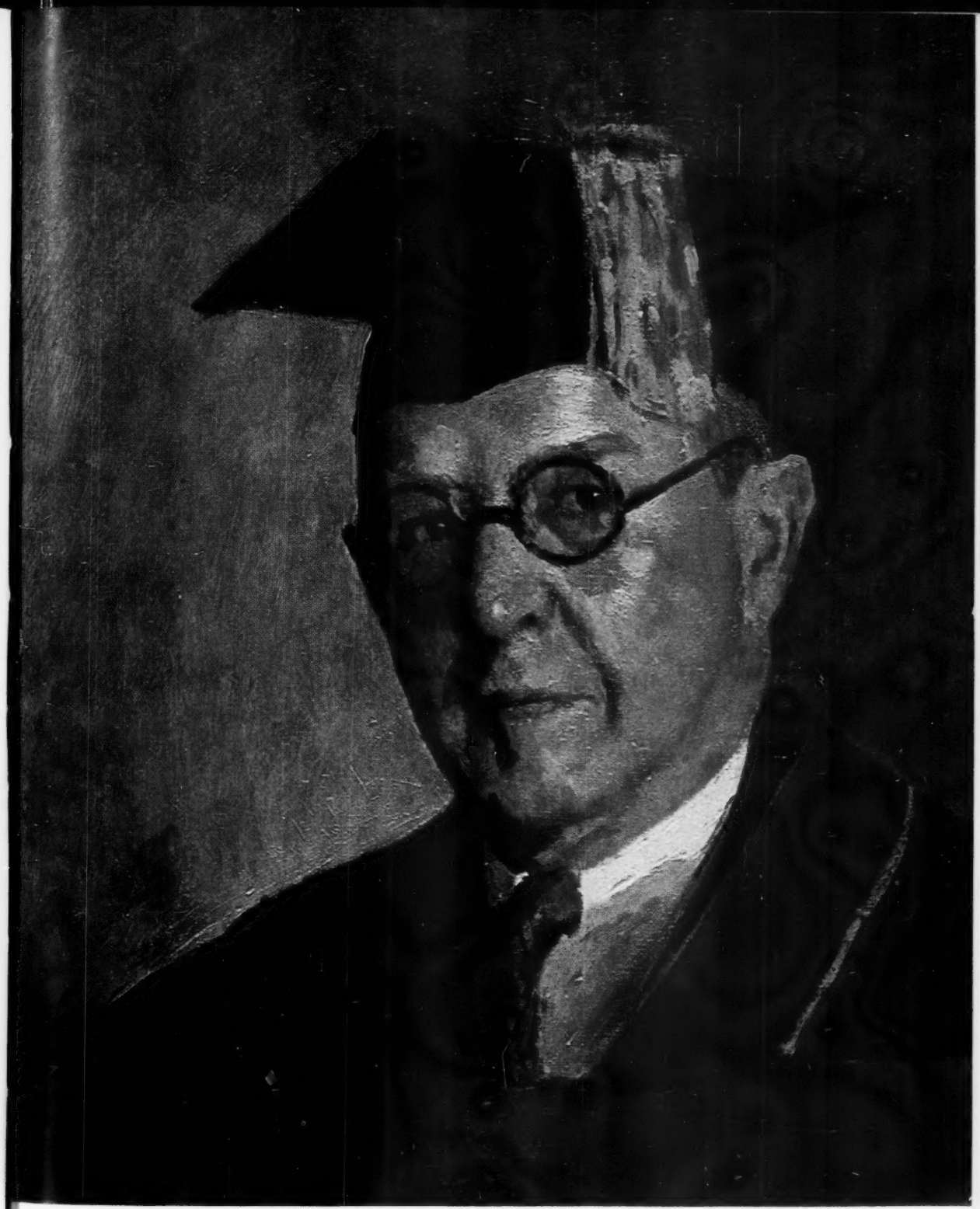

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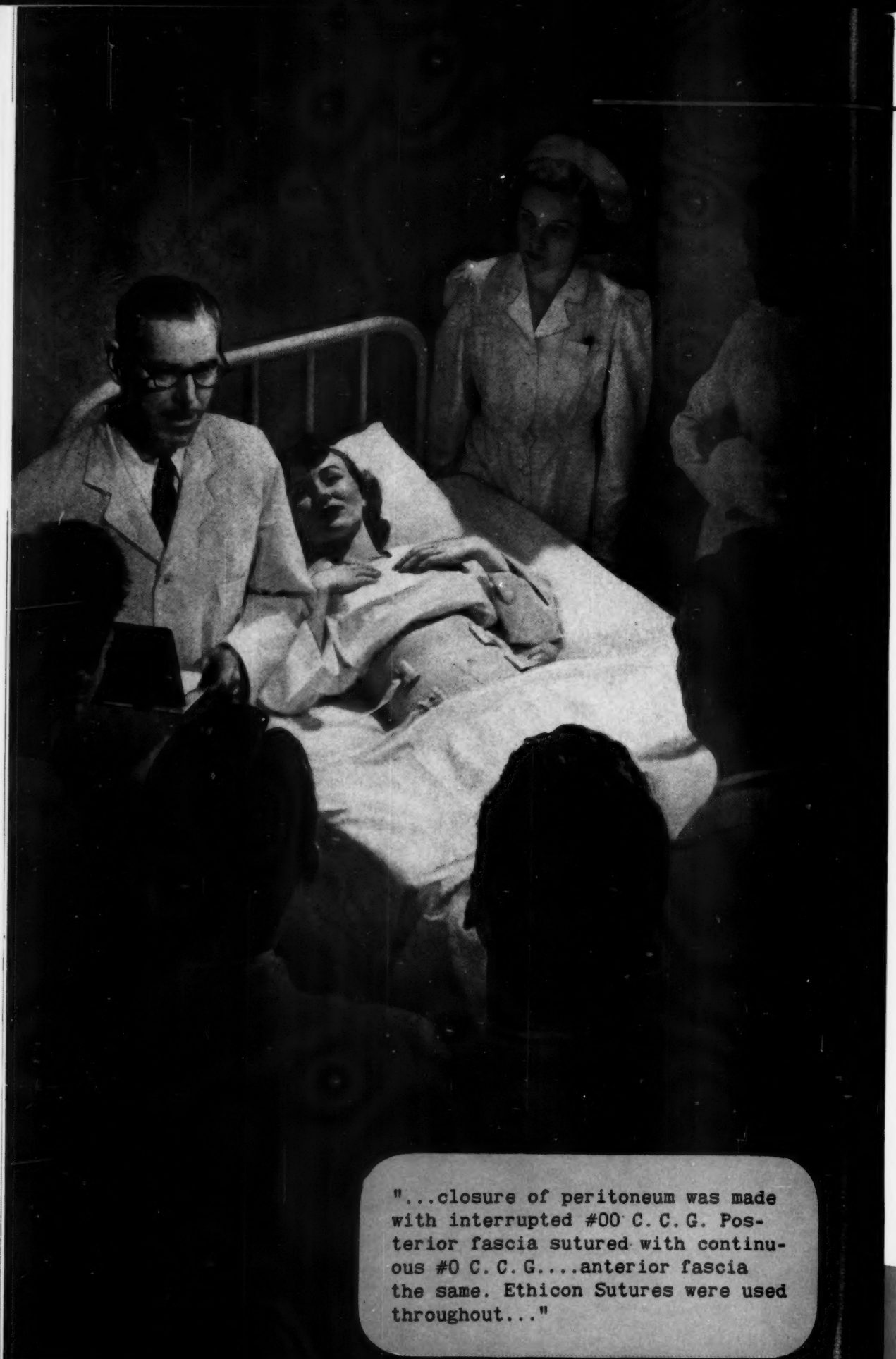
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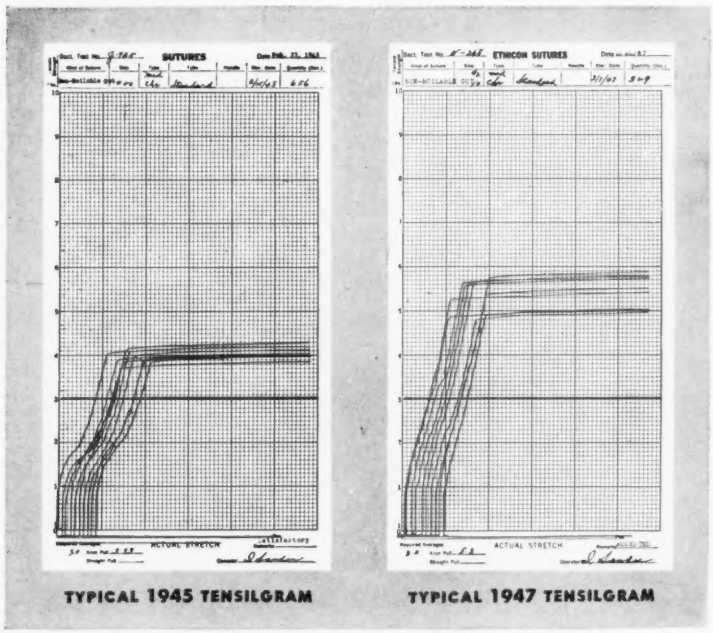

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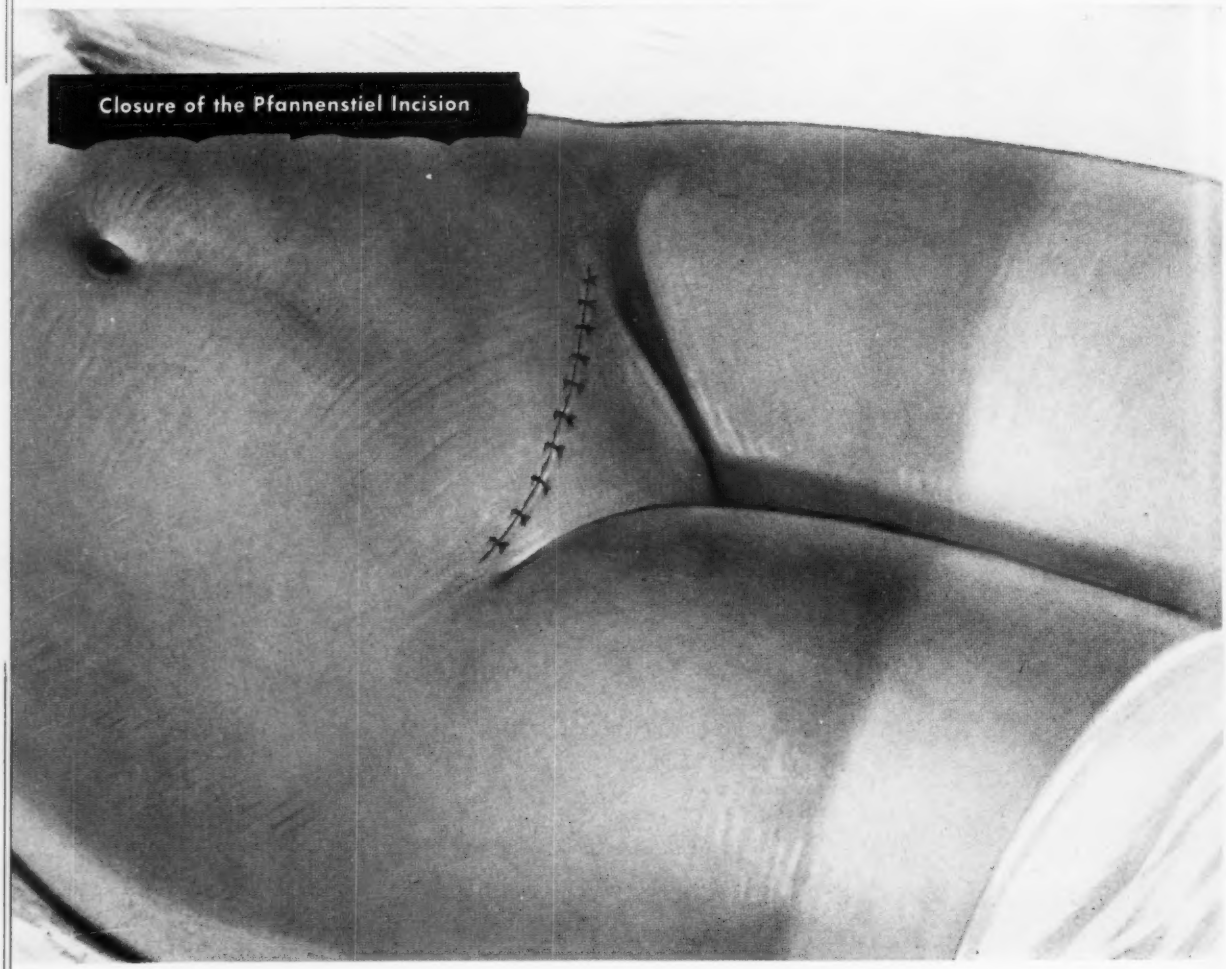

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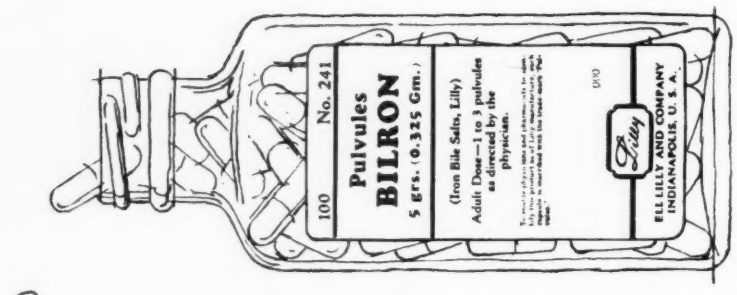

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ETIOLOGY OF ACUTE PANCREATITIS

AN EXPERIMENTAL STUDY

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ACUTE pancreatitis is characterized by the dramatic onset of abdominal pain, nausea, vomiting, and collapse, even to the state of shock. In rare cases, abdominal trauma, rupture of an aneurysm, or infections are responsible, but in the large majority the etiology is not definitely established.

The pathology of acute pancreatitis varies considerably. Although some have regarded the degrees of involvement as different disease entities, it is probable that they are stages of the same process. In the cases that come to autopsy the inflammatory process is usually patchy. Some parts of the gland show destruction of the lobules, others show necrosis of a few acini around the periphery of the lobules, and still others show normal lobules with inflammatory cells in the interlobular septa. Fat necrosis is irregular in its distribution. When the walls of blood vessels are involved one finds hemorrhagic areas intermingled with the areas of fat necrosis and parenchymal destruction. At a later stage, when the pathology has had time to develop, one finds degrees of inflammation ranging from a few leucocytes to abscess formation.

In the American literature Fitz, 1889,[2] is generally credited with first describing the pathology of acute pancreatitis; whereas Balser[4] gave a detailed account of a case in 1882. In 1901, Opie[5] performed an autopsy on a patient who had succumbed to acute pancreatitis, and found a stone impacted in the ampulla of Vater. From this evidence he postulated that acute pancreatitis was caused by an impaction of a stone at the ampulla of Vater, thus leading to a retrograde flow of bile along the pancreatic tree. This observation has led to a great deal of investigative work which aimed to prove or disprove the common channel theory. The evidence has been summarized and discussed by Ravdin and Johnston.[9] We will review only a few of the salient points in this argument.

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From 200 consecutive autopsies, Mann and Giordano[8] concluded that a common channel between the two ducts could be produced by a stone at the ampulla in only 3.5 per cent of cases. Rienhoff and Pickrell[26] studied the bile and pancreatic duct systems in 250 autopsies and concluded that a common channel could be produced through blockage of the ampulla in 17 per cent of the patients. Mann and Giordano paid particular attention to the distribution of the circular muscle fibers constituting the sphincter of Oddi. They found on serial section that this muscle group extends along both the pancreatic and bile ducts—a finding that was confirmed by Boyden.[32] Should contraction of the muscles occur so as to occlude the ampulla, the two ducts would be shut off from each other and a common channel would be impossible. Cameron and Noble[20] found that in 66 per cent of 100 routine autopsy specimens, regurgitation of bile along the pancreatic ducts was possible. This work, however, fails to consider the factor of muscle contraction or spasm. On the basis of anatomic evidence alone one must conclude that in the majority of cases, a retrograde flow of bile does not cause acute pancreatitis.

Cases of acute pancreatitis have been reported in which the common channel theory was not an acceptable explanation. Outstanding in this group are the ones which describe acute pancreatitis in the region of the gland which is drained entirely by the duct of Santorini.[10-13] This duct has a separate opening in the duodenum quite apart from the bile duct. A retrograde flow of bile will not explain the disease in these cases. In another group of cases where death followed acute pancreatitis, the duct of Wirsung opened separately from the common duct into the duodenum.[8, 10, 14, 27] Mann and Giordano[8] studied eleven cases of acute pancreatitis at autopsy and in none of them was a common channel possible from an obstruction at the ampulla of Vater.

More recently, radiographic studies have been made in patients with a T tube in the common duct. Ravdin and Johnston[9] reviewed the evidence thus obtained and found that in 20 per cent of cases there is a free flow of bile into the pancreatic tree. Such cases do not develop acute pancreatitis even though bile enters the pancreatic duct. It is quite reasonable to assume that in the patients where a common channel is possible between the two ducts there is a free flow of bile into the pancreatic duct and vice versa depending upon the relative pressures in the two systems.

Finally, there is the criticism of the animal experiments which produced acute pancreatitis by a retrograde injection of bile along the pancreatic duct. Rich and Duff[14] showed that the pressures used for such retrograde injection were unphysiologic. They used 2 c.c. of India ink for retrograde injection and noted that even such small amounts ruptured the ducts and led to an extrusion of the carbon particles into pancreatic tissue. In experiments with retrograde injection of bile into the pancreatic duct, as much as 5 to 10 c.c. was used.[15] Archibald[15] used pressures up to 500 mm. Hg, whereas physiologic pressures do not exceed 375 mm. of bile.[8] We must conclude that in all of such experiments, rupture of the pancreatic ducts is a complicating factor with its resultant injury to the gland and release of pancreatic juice into the intercellular

spaces. In confirmation of this criticism we have the observation by Mann and Giordano that pancreatitis cannot be produced by retrograde injection of bile if the pressure exerted is within physiologic limits.

The experiments in which a common channel was formed between the bile and pancreatic ducts are open to a further criticism for they involve obstruction of the pancreatic duct. What would happen under the identical experimental conditions if only the pancreatic ducts were tied has not been studied as a control. Furthermore, it is not reported in detail whether animals used for the experiments were fed or starved—a most important consideration in dealing with the pancreas.

A theory regarding the etiology of acute pancreatitis which held the attention of clinicians some years ago was the spread of infection from the gall bladder through the retrograde flow of lymph. A review of the evidence on this subject seems scarcely necessary, for this concept does not bear careful scrutiny. Ravdin and Johnston have summarized the evidence against its validity.

One of the considerations which has made the common channel theory and the resultant flow of bile into the pancreatic tree so attractive has been the activation of trypsin produced by bile. Some explanation had to be offered for the activation of trypsinogen into trypsin in order to account for the autodigestion of the pancreas in acute pancreatitis. That bile is not essential for this activation is apparent when one considers the great number of agents that will activate trypsinogen. According to Babkin[16b] Hcl, bile, calcium salts, tissue extracts, bacteria and other substances will activate trypsinogen. If the pancreatic juice is expressed into the pancreatic tissues, trypsinogen will therefore be activated immediately by the calcium ions. Lipase and amylase are secreted in active form and can, therefore, act at once when they reach the tissue spaces. Dragstedt, Haymond, and Ellis[28] argued against the capability of pancreatic juice digesting pancreatic tissue. They sutured the tail of the pancreas into the duodenal wall so that the active duodenal contents could act on it. Since no digestion occurred they concluded that pancreatic juice is harmless without the action of another factor. In acute pancreatitis the pancreatic secretion is extruded into the tissue of the gland, presumably under pressure, for it must burst the finer duct walls to reach the intercellular spaces. This is a far different set of circumstances from a mere passive contact of the juice with the surface of the gland. The photomicrographs presented by Dragstedt and associates[28] show a heavy layer of fibrous tissue on the surface of the pancreas, indicating that the duodenal contents have no direct contact with the pancreatic acini.

In the experiments of Polya,[29] acute pancreatitis was produced by retrograde injection of small amounts of pure trypsin into the pancreatic duct. The same results were obtained by Rich and Duff.[14] Both investigators failed to produce pancreatitis with an equal amount of heated or inactivated trypsin. Once the pancreatic juice reaches the tissue spaces it begins to produce necrosis and inflammation. The main point is, therefore, to determine how the pancreatic juice reaches the tissues.

We find in the literature a number of observations pointing to the etiology of acute pancreatitis. Coffey[17] produced acute pancreatic inflammation and fat necrosis in dogs by ligating the main pancreatic duct and administering pilocarpine. His experiments are open to the criticism that he avulsed the lesser pancreatic duct thereby allowing pancreatic juice free access to the peritoneal cavity. Wangensteen and associates[18] also produced fat necrosis and inflammation of the pancreas in cats by ligating the major duct and giving pilocarpine. One of us (R.L., unpublished observations) noted in some experiments performed with Florey[19] that there was an acute inflammatory reaction in the pancreas of cats after prolonged stimulation of the vagus nerves with the pancreatic ducts ligated. Hess[33] found that ligation of the pancreatic ducts in dogs at the height of digestion produced acute pancreatitis and fat necrosis. This did not occur if the ducts were ligated in starved animals. Wangensteen and his co-workers[18] were able to produce acute pancreatitis by ligating the ampulla of Vater in cats and administering fatty meals. They did not perform the same experiment with simple ligation of the pancreatic duct as a control.

Wangensteen and associates,[18] after an exhaustive study of experimental pancreatitis in cats, concluded that this disease represents not one but perhaps several factors working in combination. These experiments all have two factors in common—(1) obstruction to the pancreatic ducts in the presence of a (2) secreting or stimulated gland. We believe that these two factors are responsible for most cases of acute pancreatitis. Rich and Duff[14] came to a similar conclusion from their study of human autopsy material. The present study is experimental confirmation of the evidence which these authors collected from human material. It seems unfortunate that Rich and Duff[14] should have been quoted so widely in the literature as explaining acute pancreatitis by obstruction due to proliferation of ductal epithelium. A careful reading of their paper shows that their most important idea is blockage of a secreting gland, and that ductal hyperplasia is only one form of such blockage.

EXPERIMENTAL

It was the object of these experiments first to obstruct the pancreatic ducts and then to stimulate the gland by various means. Feeding is the natural stimulant of the pancreas. Any parasympathomimetic agent will evoke secretion. Vagal stimulation, acetylcholine, pilocarpine, and eserine are such agents. There is also a hormonal stimulation peculiar to the pancreas—secretin. This agent is elaborated in the duodenum by the action of acid on the duodenal mucosa. Secretin yields a secretion poor in anzymes and quite watery, whereas vagal stimulation elicits a thick secretion quite rich in enzymes. It seems likely that the enzymes are formed in the pancreas during the early phase of digestion through vagal stimulation and then are "washed out" by the action of secretin which is formed and absorbed as the stomach contents reach the duodenum. The simplest way to be certain of a secretion rich in enzymes is to feed the animal.[14]

Many experiments were performed in which the animals were fed two hours before ligation of the ducts. The animals were then given secretin,* pilocarpine, or acetylcholine and eserine. It was found that the degree of pancreatitis was not increased over that obtained by feeding and tying the ducts without further stimulation. In subsequent experiments starved animals were used when these agents were administered.

Medium-sized cats of both sexes were used. Under ether anesthesia a midline upper abdominal incision was made using sterile precautions. By very gentle dissection the greater and lesser pancreatic ducts were located. Ligation of the greater duct is fairly simple. It enters the duodenum alongside the common bile duct and is usually 1 to 2 mm. in diameter. The lesser duct may be rather elusive, as it is covered by glandular substance and may be only the size of a fine thread. It is best approached from the side opposite to the entrance of the large duct. At first we were unable to locate this duct in many animals and our results were quite variable. After perfecting the operative technique we were able to find the lesser duct in all of the cats. We include in our experimental reports only those animals where both greater and lesser ducts were found.

Two sets of experiments were performed with each method of stimulating the pancreas. In one group the ducts were located but not tied; in the other a silk ligature was placed around both ducts. In this way the element of surgical trauma was eliminated as a source of pancreatic inflammation.

Secretin was given intravenously using a total of 10 to 20 units in divided doses during the experimental period. Pilocarpine was administered subcutaneously in 0.6 mg. doses, repeated twice a day throughout the experiment. One milligram of acetyl-beta-methylcholine was combined with 0.6 to 2.4 mg. eserine for each dose. This also was repeated twice a day. When the animals were fed they were given chopped liver and milk. In a separate group of experiments the ducts were tied and 100 c.c. of 20 per cent cream was administered by stomach tube immediately after laparotomy. This was repeated under light ether narcosis twice a day until the animal was sacrificed.

The cats were observed for varying periods. The best time for sacrifice seemed to be twenty-four hours or forty-eight hours, and most of them were killed by ether inhalation at the end of this time. Because the operative trauma might give an inflammatory reaction, sections of the gland were taken through the tail of the pancreas at a considerable distance from the ligation. The tissues were fixed in Zenker's solution and stained with phloxine-methylene blue.

In all animals that had a simple laparotomy with exposure of the ducts, but without ligation, there was no fat necrosis seen. Normal pancreas was found on microscopic section (Fig. 1). The same results were obtained when the ducts were tied in starved animals and no stimulant administered (Fig. 2). Negative results were obtained when the animals were fed, or given pilocarpine, secretin, or acetylcholine and physostigmine with the ducts intact. When the

*Two preparations of secretin were used; one Secretin Astra and the other Secretin furnished by the Wyeth Corporation. Both preparations seemed to have the same amount of activity.

Fig. 1.

Fig. 2.

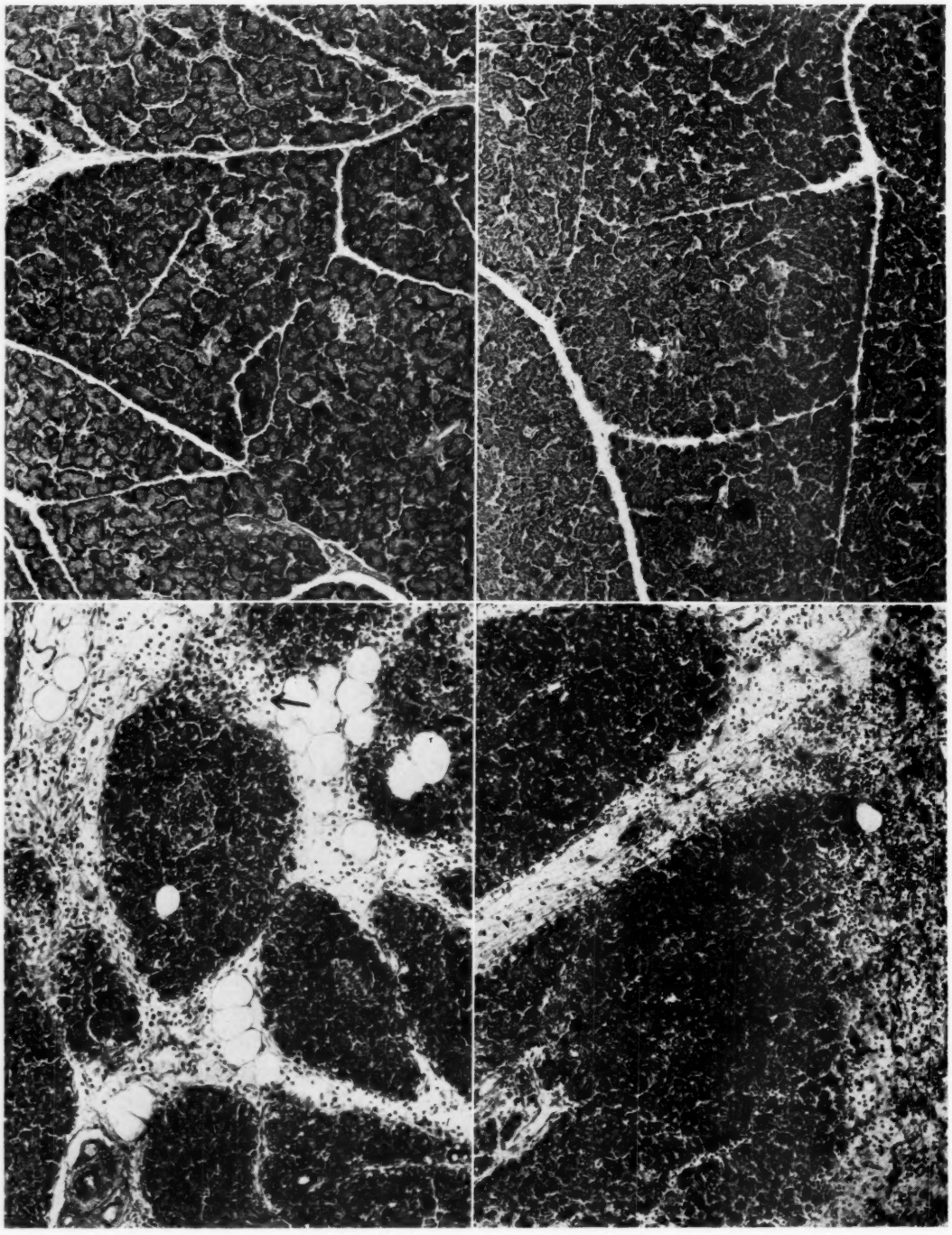

Fig. 3.

Fig. 4.

Fig. 1.—Pancreas of cat that was starved twenty-four hours and then had laparotomy with exposure but not ligation of the pancreatic ducts. Sacrificed forty-eight hours after laparotomy.

Fig. 2.—Pancreas from cat that had been starved twenty-four hours before the experiment; ducts ligated; no pancreatic stimulation given. Sacrificed forty-eight hours after ligation of the ducts.

Fig. 3.—First stage of pancreatic inflammation as described in text with widening of interlobular septa and acute inflammatory reaction of mild degree. The acini are intact except for the region indicated by the arrow. Here there is some disintegration of the lobule.

Fig. 4.—Stages 2 and 3 with more intense inflammatory reaction between the lobules and destruction of acini along the whole length of the lobule. There are also some inflammatory cells seen in the substance of the lobule. Sacrificed forty-eight hours after ligation of ducts in a starving cat that was given pilocarpine.

TABLE I

NO. OF CATS	DUCTS	FED OR STARVED	DURATION	STIMULATION	FAT NECROSIS	GRADE 1	2	3	4
4	Exposed not tied	Starved	48 hr.	None	0	0			
3	Exposed not tied	Fed	Died at operation	None	0	0			
4	Tied	Starved	48 hr.	None	0	0			
2	Exposed not tied	Fed	9 days	Secretin	0	0			
4	Tied	Starved	4 days	Secretin	3	1	1	1	1
2	Tied	Starved	4 days	A. choline secretin	2			2	
6	Tied	Fed	24 hr.	Secretin	5	1		2	3
8	Tied	Fed	48 hr.	A. choline eserine	8	3		2	3
6	Tied	Starved	48 hr.	Pilocarpine	6	2	1	1	2
2	Exposed not tied	Starved	48 hr.	Pilocarpine	0	0			
17	Tied	Fed	48 hr. to 6 days	None	14	2	4	6	5
6	Tied	Fed by tube after operation	48 hr.	None	2		3	2	1

ducts were tied and the animals were fed, or the stimulants administered, gross fat necrosis was found in the majority of the animals as noted in Table I. The amount of fat necrosis varied greatly. In the least involved a few scattered areas were found along the mesentery of the pancreas and omentum. The animals with greatest involvement showed a wide distribution of fat necrosis in the omentum, and throughout the mesentery of the intestines and stomach. Between these two extremes were all degrees of this type of lesion. The pancreas from animals that had ligation of the ducts and stimulation by feeding, secretin, or other drugs showed on microscopic section an inflammatory reaction which varied widely in degree. This inflammatory process seems to lend itself to classification in four categories.

1. The mildest degree of pancreatitis noted is a widening of the septa with presence of inflammatory cells and exudate in the septa. The pancreatic tissue appears quite normal (Fig. 3).

2. The next stage shows a more extensive inflammation in the septa and beginning invasion of the lobules by inflammatory cells (Fig. 4).

3. In addition to these changes, the third stage reveals a beginning destruction of acini. This is most pronounced at the periphery of the lobule, but occasionally one finds local areas of acinar destruction inside the lobule (Fig. 4).

4. At this stage the entire lobule is undergoing dissolution and is replaced by inflammatory cells (Figs. 5 and 6).

If the experiments are continued over forty-eight hours, one finds the inflammatory process proceeding to organization with fibroblastic proliferation. The amount of fibrous tissue laid down is commensurate with the previous destruction and since it follows the lines of the previous inflammation one sees the greatest amount of fibrous tissue around the periphery of the lobules.

Fig. 5. Fig. 6.

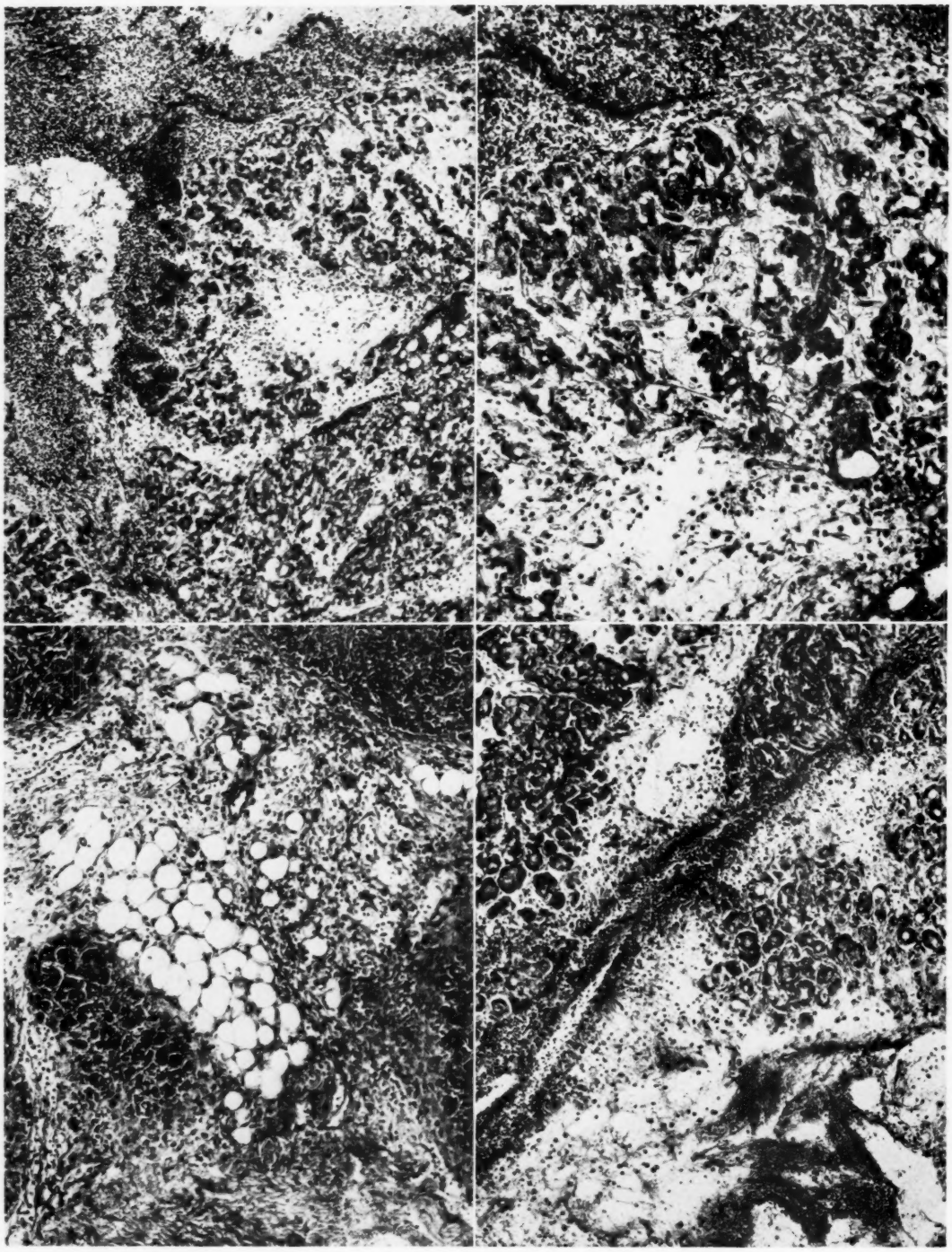

Fig. 7. Fig. 8.

Fig. 5.—Stage 4, where the entire lobule is undergoing dissolution and acute inflammatory reaction is everywhere to be seen. Animal fed, ducts ligated, and animal sacrificed forty-eight hours later.

Fig. 6.—High-power view of Fig. 5.

Fig. 7.—Pancreatic destruction and local fat necrosis forty-eight hours after ligation of the ducts in a starving animal and giving secretin intravenously.

Fig. 8.—Grade 4 involvement of pancreas. Animal starved, given acetylcholine and eserine just before ligation of the ducts. Intravenous secretin administered after ducts were tied. Sacrificed forty-eight hours after operation.

In the experiments with pilocarpine, acetylcholine, and eserine, where starved animal was used, a few areas of fat necrosis were found and scattered areas of acute inflammation with destruction of pancreatic lobules were observed. Otherwise the gland appeared quite normal. This contrasts sharply with the fed animals and those that were given secretin. In these there was some inflammation through the entire gland with scattered foci of marked damage or complete destruction.

Vascular lesions were not found in most of our sections, but there were patchy areas of hemorrhage mingled with the inflammatory reaction in some of the glands. These areas were noted only in those with grade 3 and 4 involvement. Some sections reveal damage to the walls of the blood vessels with destruction of the muscularis as noted by Rich and Duff.[14] However, we did not obtain extensive vascular damage as seen in human patients.

Failure to obtain gangrenous pancreatitis and extensive vascular damage puzzled us, but we believe that a probable explanation for this failure is the anatomy of the cat's pancreas. The gland in the cat is thin and relatively free as compared with the human pancreas, which lies against the posterior wall of the lesser peritoneal cavity in a fairly snug connective tissue compartment. In the cat any internal pressure of secretion can force the juice to the surface quite readily; whereas, in the human being, the thickness of the gland and its dense capsule allow the extruded secretion to remain longer in the gland substance before reaching the surface. The resultant autodigestion would, therefore, be more intense in the human being. There is very little fatty tissue within the cat's pancreas, whereas the human being presents many islands of fat cells. We found that the worst damage to the pancreatic tissue was usually adjacent to an area of fat necrosis on the surface of the gland.

DISCUSSION

One can produce inflammatory lesions of the pancreas and fat necrosis by ligating the ducts in starving animals and then stimulating the gland by artificial means. But the most constant and extensive inflammatory changes follow ligation of the ducts at the height of digestion, or about two hours after the animal has eaten.

Are the changes we have produced necessarily the same as those of acute pancreatitis in human beings? The patchy distribution of severe lesions is quite characteristic of acute pancreatitis and the finding of normal lobules alongside those that are badly damaged is a common one. Eggers[34] reported a very interesting fatal case of acute pancreatitis in which there were many normal areas of gland tissue. The necrosis and inflammation proceeded along the interlobular septa and attacked the periphery of the lobules first. This same picture was found in our experiments. Reconstructing the sequence of events on the basis of the four degrees of pancreatic involvement noted in our animals, we have something of this order. The pancreas is secreting actively, and suddenly the outlets are obstructed. The pancreatic juice ruptures the ducts which run along the interlobular septa and initiate an inflammatory

reaction as the result of tissue damage. If the secretory pressure and enzymatic concentration are not great, the process may end at this stage, but if the secretory pressure is high or the enzymatic content of the juice is high, the autodigestion of tissue proceeds to an attack of the acini on the periphery of the lobules as well as a penetration into the lobules. The patchy distribution of severe lesions can readily be explained by the location of the ductal rupture. Certainly the ducts will not rupture throughout their entire length, but rather in a few places. Wherever they rupture, the pancreatic juices will escape and produce local damage of an intensity commensurate with the volume and enzyme content of the extruded secretion.

In the light of these experiments most of the experimental and clinical facts can be rationalized. The occurrence of acute pancreatitis after a heavy meal at the height of digestion has been noted by clinicians for years. This is the time when the pancreatic juice contains the greatest concentration of enzymes.[14] An obstruction of the gland at this time will thus cause the greatest autodigestion and tissue damage.

What causes the obstruction to the pancreatic ducts in the human being? Mechanical factors such as stones and ascaris at the ampulla have been summarized by Ravdin and Johnston.[9] Archibald argued that in the absence of such elements, edema and spasm of the sphincter of Oddi could produce obstruction. He observed this spasm in cats. With the pressure in the biliary tree kept constant by a manometer, the fluid was seen to enter the duodenum at intervals rather than as a constant flow. In the light of clinical studies with tubes in the common duct, this seems quite reasonable. Best and Hicken[21] found marked elevation in the intraductal pressure after cholecystectomy, and they found that spasm of the sphincter caused complete although temporary obstruction. McGowan, Butsch, and Walters[22] reported a marked increase in bile pressure along the common duct after morphine sulfate had been given, indicating mechanical blockage by spasm. This was relieved by glyceryl trinitrate. Ivy and Sandbloom[30] showed that blockage of the biliary passages by spasm is possible in normal human subjects and that magnesium sulfate placed in the duodenum relieves the stoppage in a few minutes. Any blockage of the bile passages at the ampulla will also obstruct pancreatic secretion. That spasm of the smooth muscle about the ducts may be responsible in acute pancreatitis is suggested by Elman,[23] who noted that glyceryl trinitrate placed under the tongue shortly after an acute attack of pancreatitis has begun may lead to a dramatic abortion of the attack.

This relief by glyceryl trinitrate may not be due entirely to relief of spasm in the sphincter of Oddi. It may also relieve spasm of the pancreatic ducts. In this connection von Anrep[24] published some significant but seldom quoted experiments on pancreatic secretion. This investigator studied the simultaneous changes in the size of the pancreas and secretory rate during vagal stimulation. During early stimulation of the vagus the pancreas increased in size. At first there was no secretion of juice, but as the stimulation progressed the juice began to flow slowly and to increase gradually until it was

running at a steady rate through the cannula. Simultaneously with the flow went a subsidence of the glandular edema, and at the height of secretion the gland had returned to its original size.

Edema of the pancreas under normal conditions is a temporary stage associated with vagal stimulation. This edema may be due to secretion retained in the cells or to spasm of the ducts. This normal reflex, when pushed beyond physiologic limits, could provide complete obstruction to pancreatic secretion being elaborated in the gland and a resultant backflow into the tissue spaces.

A further possible mechanism of obstruction is edema of the duodenal mucosa. In patients who have overindulged in alcoholic beverages, hyperemia and edema of the duodenal mucosa are common findings. The opening of the bile and pancreatic ducts into the duodenum are very small and could be closed temporarily by such swelling. Archibald[15] demonstrated this clearly in his experimental work. Alcohol is a stimulant of both gastric and pancreatic secretion, and any obstruction of the pancreatic ducts occurring after ingestion of this agent would lead to the development of pancreatitis. The relation of alcoholism to clinical pancreatitis has been reported by Myers and Keefer.[35]

The cause of obstruction in patients with gallstones who do not have a stone impacted at the ampulla is not entirely clear. The most obvious explanation at hand is that in such patients spasm of the pancreatic ducts or the sphincter of Oddi is much more readily produced than in normal patients. Wangensteen and associates[18] furnish some suggestive evidence for this view. These investigators were unable to produce mechanical blockage of the bile and pancreatic ducts by spasm in normal cats. They were, however, successful in producing obstruction by spasm in one animal that had an established infection of the gall bladder.

The degrees of pancreatitis seen clinically depend on many variables. The combination of obstruction and secretion may occur at different stages of the digestive process. The obstruction may be subacute or total. It may occur at the height of secretion or at the end.

For minimal pancreatitis or the edema as described by Elman[23] we can assume either temporary obstruction or total obstruction with minimal secretion. In more advanced stages, the obstruction is prolonged. When lesions to the blood vessels occur, the obstruction has not only been of considerable duration but also the secretion has a goodly amount of proteolytic enzymes. The rare case of pancreatic apoplexy occurs when a sudden complete obstruction develops to a secretion that is copious and rich in digestive enzymes.

SUMMARY

Experimental and clinical evidence concerning the etiology of acute pancreatitis has been reviewed.

Experiments have been reported of acute pancreatic inflammation and fat necrosis produced by tying the pancreatic ducts and stimulating the pancreas. Stimulation of the pancreas has been produced by (1) feeding, (2) acetylcholine and eserine, (3) pilocarpine, (4) stimulation of the vagus, and

(5) secretin. In a high percentage of these experiments, combining obstruction of the pancreatic ducts and stimulation of the gland, acute inflammation of the pancreas and fat necrosis resulted. The most extensive and consistent damage resulted when the animals were fed two hours before ligation of the pancreatic ducts. The different mechanisms that can produce obstruction of pancreatic secretion have been discussed.

The clinical and experimental evidence has been correlated to explain the varying degree of acute pancreatitis seen at operation or autopsy.

CONCLUSION

Acute pancreatitis is the result of ductal obstruction in an actively secreting pancreas.

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A COMBINED ABDOMINOTHORACIC INCISION PARTICULARLY ADAPTED FOR USE IN TOTAL GASTRECTOMY AND ESOPHAGOGASTRECTOMY

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IN AN attempt to extirpate cancer of the stomach, surgeons have become progressively bolder and more aggressive. The operation of total gastrectomy, which a generation ago carried almost a prohibitive mortality, has today become a relatively common procedure in good surgical clinics. It was first successfully performed in 1897 by Schlatter,[12] and in 1898 the first successful total gastrectomy in America was performed in San Francisco by MacDonald.[8]

In 1929 Finney and Rienhoff[2] reported sixty-two cases collected from the literature, to which they added five cases of their own. Of these sixty-seven cases there were thirty-six deaths, a mortality rate of 53.8 per cent. Recently Longmire[7] recorded his experience with twenty consecutive transabdominal total gastrectomies in which he reported two deaths, a 10 per cent mortality rate. In this clinic since Jan. 1, 1947, fifteen total gastrectomies have been performed with one death. These operations have been done by several members of the surgical staff of the University Hospitals. Some of these operations were further complicated by requiring concomitant resection of the lower esophagus, spleen, pancreas, colon, and other neighboring organs.

Although surgeons latterly are reporting mortality rates in the area of 10 per cent for transabdominal total gastrectomy, the transthoracic, transdiaphragmatic approach for total gastrectomy and resection of the lower esophagus with esophagojejunostomy is still associated with a formidable mortality rate. The first successful operation of this type was reported in 1942 by Meyer.[10] Later Sweet,[14] in 1944, reported six deaths in thirteen patients operated upon. In summarizing his series in 1945[15] he reported seven deaths in a series of eighteen cases, a mortality rate of 38.8 per cent.

Since the major portion of a total gastrectomy with resection of the lower esophagus involves principally in abdominal dissection, and yet the esophagojejunal anastomosis must be performed in the chest, it was felt that a combined abdominothoracic incision would offer the best approach for these cases. Especially is this true for those surgeons who by virtue of training and experience find it easier to work principally in the abdomen, on familiar ground, and from a customary position and stance at the operating table. A description of such an incision and our experience with it is the purpose of this communication. This incision has been employed principally in cases for total gastrectomy with resection of the lower esophagus. One additional patient is included in this series in whom the combined abdominothoracic incision was successfully utilized in the repair of a large paraesophageal hernia.

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Before the physiology of respiration was adequately understood, surgeons were loath to open the pleural cavity. Marwedel,[9] in 1903, in an effort to remove the barrier of the costal margin in exposing the area of the esophageal hiatus, devised a complicated incision cutting the costal cartilages and reflecting the costal margin laterally as a flap without entering the pleural cavity.

Kirschner,[6] in 1920, described a combined abdominothoracic incision which he used in four cases which is quite similar in principle to the present-day combined abdominothoracic approach. His incision, with the patient in the lateral decubitus position, began at the boundary of the proximal and middle third of a line connecting the xiphoid process and the umbilicus, extended obliquely downward and laterally to the left costal margin, then across the eighth costal cartilage and out into the seventh interspace to the angle of the scapula. The diaphragm was then incised from the costal margin to the esophageal hiatus.

Ohsawa,[11] in 1933, used a combined abdominothoracic incision with a vertical abdominal component for resection of high gastric lesions. Humphreys,[5] in 1946, utilized a similar combined incision but with a transverse abdominal component for resection of high gastric and low esophageal neoplasms. Garlock[3] also utilized a combined abdominothoracic incision for partial esophagogastrectomy. Harper[4] wrote of employing a combined abdominothoracic incision in approaching the organs of the upper abdomen, entering the thorax through the bed of the resected ninth rib, and employing only a short abdominal component to the incision. Recently the use of a combined abdominothoracic incision for difficult splenectomies was described by Carter.[1] In none of the previous reports, however, has the incision been devised and utilized to provide for principally an abdominal dissection, with the idea of extending the horizon of total gastrectomy to include removal of additional segments of the lower esophagus.

TECHNIQUE

The patient is positioned supine on the operating table with a sandbag under the left side, producing about a 10 degree elevation from the horizontal. A kidney brace is fastened on the right side of the table to hold the patient in position when the table is rotated later in the operation. The skin incision (Fig. 1) begins near the lateral edge of the right rectus muscle slightly above the umbilicus and extends horizontally and upward to the left costal margin where the eighth costal cartilage crosses the seventh interspace. The incision is carried through the abdominal wall into the peritoneal cavity. Abdominal exploration is carried out at this time and operability of the lesion determined. If a decision to proceed with resection is made at this point, the operating table is rotated, elevating further the left side of the patient to 20 to 25 degrees from the horizontal. The eighth costal cartilage is cut and the incision is extended into the pleural cavity through the seventh interspace as far as the mid- or posterior axillary line. If total gastrectomy alone with infradiaphragmatic esophagojejunostomy is to be performed, incision of the diaphragm about halfway from the costal margin to the esophageal hiatus is carried out (Fig.

2). If an additional segment of the esophagus is to be resected, the diaphragmatic incision is continued to the esophageal hiatus (Fig. 3). The left phrenic nerve, as it passes along the lateral surface of the pericardium, is crushed early in the operation. This reduces motion of the diaphragm, and aids in its subsequent repair and healing.

Dissection of the stomach particularly around the cardia and high along the lesser curvature in the area of the left gastric artery is markedly facilitated by removing the barrier of the costal margin. The abdominal portion of the operation, dissection of the lower stomach, transection of the duodenum, and duodenal closure are readily accomplished. Mobilization of a jejunal loop for esophageal anastomosis is facilitated, and if necessary one can readily section

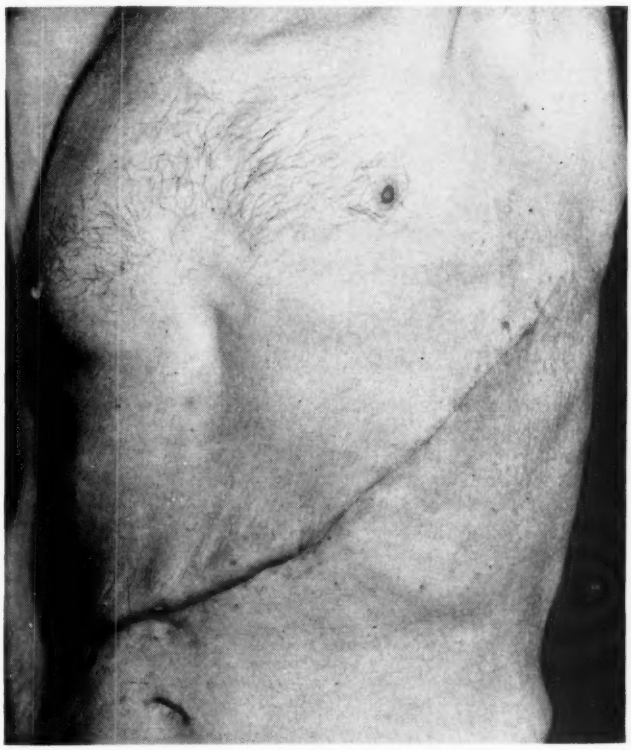

Fig. 1.—Skin incision.

one or more of the mesenteric vessels near their origin to gain added mobility of the jejunal loop as recommended by Sweet.[13] In performing the esophagojejunal anastomosis, the technique of Wangensteen[16] whereby the posterior side of the anastomosis is buttressed by two layers of interrupted silk sutures, starting along the mesenteric border of the jejunum, was regularly employed. All portions of the operation are performed without limitation of exposure. Because of the added exposure, a wider and more complete excision of lymphatic-bearing tissue can be accomplished. Although the incision opens two serous cavities, it is felt that the added exposure gained allows for a more careful dissection, preparation, and anastomosis, so that leakage and infection, which

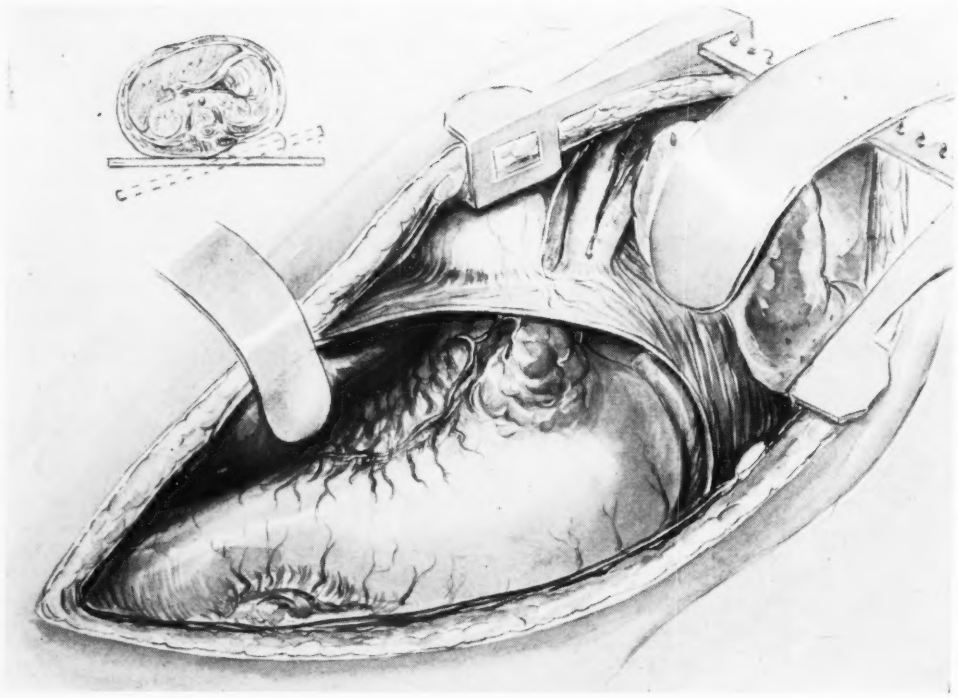

Fig. 2.—Incision opened with diaphragm incised part way to the esophageal hiatus. Insert shows position of patient on the table with a sandbag under the left side. Dotted line shows rotation of table and patient prior to making intercostal part of the incision.

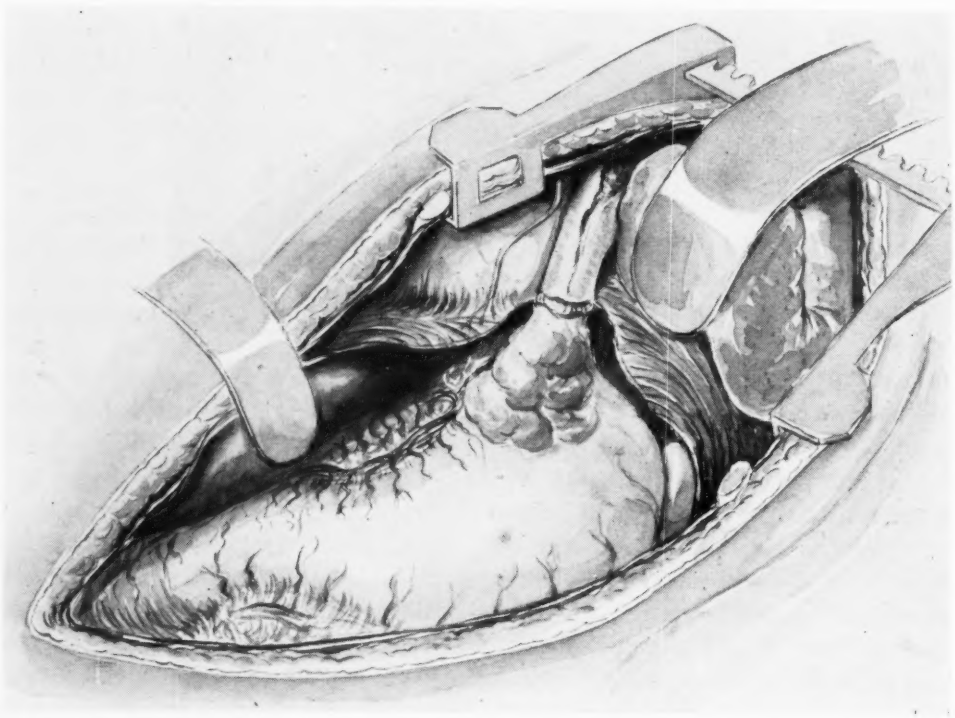

Fig. 3.—Exposure obtained with diaphragm incised to esophageal hiatus.

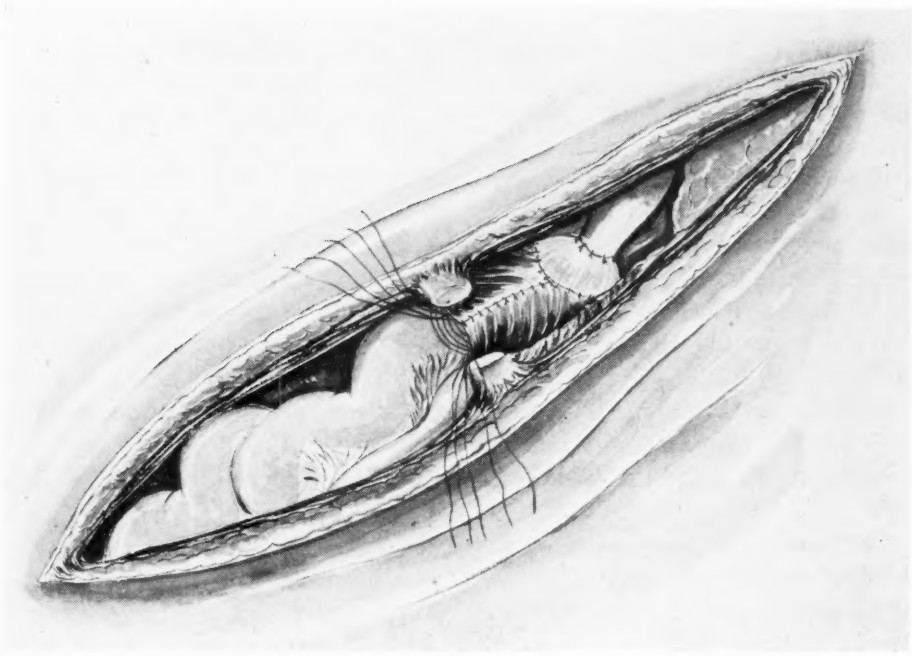

Fig. 4.—Closure of diaphragm, diaphragm sutured to jejunal loop. The most anterior sutures cannot be tied until ribs are approximated. These are placed from the peritoneal surface and tied through the abdominal part of the incision after the intercostal incision is closed.

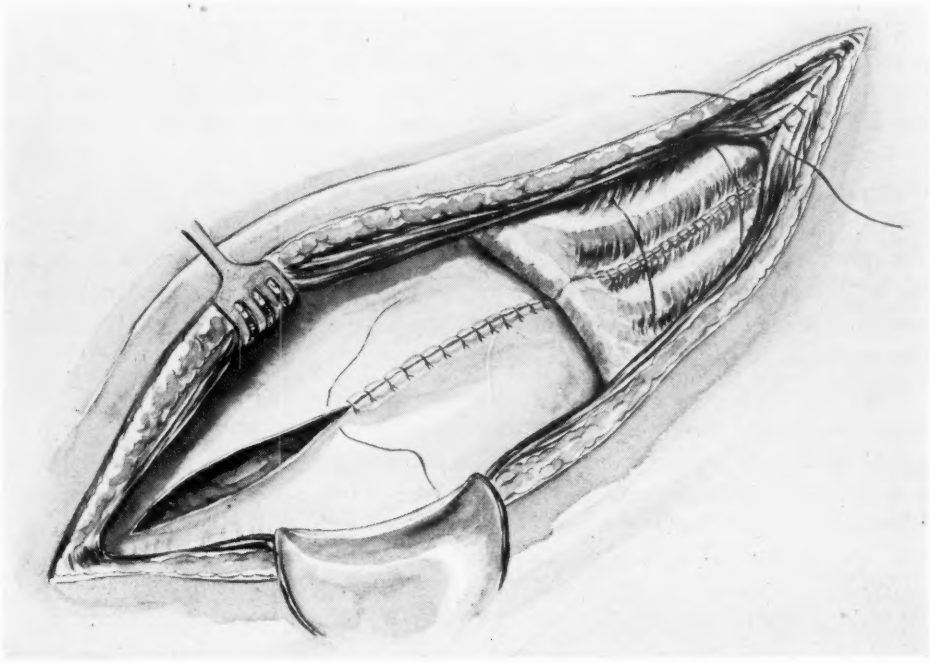

Fig. 5.—Closure of the peritoneal layer after intercostal space is closed.

has been the principal cause of fatal outcome in most reported cases, are less likely to result. Resection of an added segment of esophagus, in doing a total gastrectomy, is readily accomplished with the exposure obtained, whereas in doing a transabdominal total gastrectomy one cannot with ease sacrifice more than small segments of the esophagus.

Closure of the intercostal incision is shown in Figs. 4 and 5. The diaphragm is circumferentially sutured to the anastomotic jejunal loop to prevent any herniation of abdominal viscera into the thoracic cavity. The last few anterior sutures in the diaphragm are placed from the peritoneal surface and tied later from below after the intercostal incision has been closed. In two areas the rib above and below the incision is bared of periosteum, and perichondral sutures of 0 chromic catgut are passed around the ribs, care being taken to avoid catching the intercostal nerves. Tying of these sutures closes the intercostal space. This is further closed by interrupted 000 or 0000 silk sutures uniting the cut intercostal muscle bundles. This is reinforced by suturing the serratus anterior muscle.

The abdominal component of the incision is closed in the usual fashion, using interrupted 000 silk in the peritoneal layer and in the anterior rectus sheath. Because of the length of incision the closure takes considerable time. This can be shortened by having two closure teams working from either end toward the center.

Table I presents in summary our experience with the combined abdominothoracic incision in ten cases. In all, excellent exposure for all phases of the operation was obtained. No complications attributable to the incision were noted. In those cases in which supradiaphragmatic esophagojejunal anastomosis was performed, closed intercostal catheter drainage of the pleural cavity was employed for several days postoperatively. In the cases where infradiaphragmatic anastomosis was employed, no pleural drainage was used. One death occurred in a patient (Case 7, Table I) with recurrent carcinoma of the stomach, in whom an extensive operation was necessary to remove the neoplasm. The fatal outcome resulted from pancreatitis and peritonitis and was unrelated to the incision, which had greatly facilitated a long and difficult operation. Although the incision has been used principally for total or subtotal gastrectomy with resection of the lower esophagus, the report of one additional patient (Case 8, Table I), in whom it was successfully utilized for the repair of a large para-esophageal hernia, is also included.

Two potential disadvantages to the incision should be pointed out. First, unless the patient is rotated somewhat laterally there is a tendency for the heart to interfere with exposure in making a high esophagogjejunal anastomosis above the diaphragm. Second, with this exposure, one is limited to dissection of the esophagus below the hilus of the lung and the inferior pulmonary vein. Above this point the esophagus is posterior to these structures and cannot be adequately exposed. An esophageal anastomosis must therefore be done below the hilus of the lung. This circumstance precludes the use of this incision for resection of lesions in the mid-esophagus, and, in any event, where one suspects that more than the lower one-fourth of the esophagus is to be sacrificed. It is

TABLE I. SUMMARY OF CASES IN WHICH THE ABDOMINOTHORACIC INCISION HAS BEEN USED*

CASE	PATIENT	AGE (YR.)	DISEASE	OPERATION	POST-OPERATIVE COURSE	REMARKS
1	J. T. (U. H. No. 775120)	66	Carcinoma of upper stomach and lower esophagus	Subtotal gastrectomy and splenectomy, esophagectomy; intrathoracic esophagogastrostomy	Left hospital on 9th postoperative day	Returned to hospital on 4th day after leaving, with mesenteric thrombosis; expired
2	U. M. (U. H. No. 752635)	41	Carcinoma of upper stomach	Total gastrectomy with infradiaphragmatic esophagojejunostomy	Left hospital on 10th postoperative day	No complications
3	M. U. (U. H. No. 774874)	48	Carcinoma of upper stomach	Total gastrectomy, splenectomy; infradiaphragmatic esophagojejunostomy	Left hospital on 8th postoperative day	No complications
4	J. B. (U. H. No. 743178)	52	Gastritis, chronic hypertrophic; polycythemia vera, cholecystitis, chronic	Total gastrectomy, cholecystectomy; intrathoracic esophagojejunostomy	Left hospital on 13th postoperative day	Developed an acute postoperative psychosis; no wound complications
5	C. R. (U. H. No. 779603)	68	Carcinoma of cardia of stomach with extension to pancreas	Total gastrectomy with resection of 5 cm. of esophagus, splenectomy, pancreatectomy; esophagojejunostomy	Left hospital on 8th postoperative day	No complications
6	O. A. (U. H. No. 774827)	63	Carcinoma of cardia of stomach and esophagus	Total gastrectomy, partial esophagectomy, splenectomy; intrathoracic esophagojejunostomy	Left hospital on 10th postoperative day	No complications
7	J. D. (U. H. No. 668827)	68	Recurrent carcinoma in residual gastric pouch; gastric resection done 3 yr. previously; extension of carcinoma to transverse colon and pancreas, cyst of liver	Total gastrectomy, resection of segment of colon, jejunum, pancreas, and cyst of liver; infradiaphragmatic esophagojejunostomy	Died on 8th postoperative day	At postmortem examination suppurative pancreatitis and peritonitis were found; all anastomoses were intact
8	L. P. (U. H. No. 778100)	59	Large paraesophageal hernia; gastric bleeding	Repair of paraesophageal hernia from above; exploratory laparotomy	Left hospital on 5th postoperative day	No complications
9	M. D. (U. H. No. 747320)	83	Carcinoma of cardia of stomach and lower esophagus	Subtotal gastrectomy and esophagectomy; intrathoracic esophagogastrostomy	Left hospital on 11th postoperative day	No complications
10	I. L. (U. H. No. 782779)	72	Carcinoma of cardia of stomach and lower esophagus	Subtotal gastrectomy and esophagectomy; intrathoracic esophagogastrostomy	Uncomplicated; left hospital on 9th postoperative day	

*Acknowledgment is hereby made to Dr. O. H. Wangensteen, Dr. C. Dennis, Dr. R. L. Varco, and Dr. D. State who, in addition to the author, performed operations herein reported.

especially useful for high gastric lesions where invasion of the lower esophagus has occurred and where in addition to resection of the stomach one wishes to resect more of the esophagus than one can conveniently do by the transabdominal approach alone.

SUMMARY

A description of and an experience with a combined abdominothoracic incision especially as it relates to use in total gastrectomy and esophagogastrectomy is presented. The incision has been employed in ten instances with excellent exposure, greatly facilitating a difficult operation. No untoward effects or complications have attended its use.

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GASTRIC RESECTION: THE SCHOEMAKER-BILLROTH I OPERATION

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THE purpose of this presentation is to consider (1) the Schoemaker modification of the Billroth I operation, (2) some of the reasons why, when feasible, this operation seems better than the Hoffmeister-Polya operation in which the treatment of the stomach is the same but reconstruction of continuity of the intestine is different, and (3) the immediate results in ninety-five cases in which the Schoemaker-Billroth I technique has been used.

The Schoemaker modification of the Billroth I operation permits greater, more extensive, resection of the lesser curvature of the stomach than other similar operations. After resection it provides for closure of this portion of the open end of the stomach, thereby producing a tubelike stomach with a stoma at the greater curvature aspect. This stoma is utilized in end-to-end anastomosis to the duodenum.

In other modifications of the Billroth I operation, as well as the original operation as described by Billroth, either the entire end of the remaining portion of the stomach is utilized for the gastroduodenostomy or less extensive resection of the lesser curvature of the stomach is carried out before closing this superior portion of the site of transection. The Schoemaker modification will permit resection along the lesser curvature to the esophagus, and even slightly into the wall of this latter structure if necessary, and yet will leave sufficient stomach to perform a gastroduodenostomy.

These advantages of the Schoemaker modification of the Billroth I operation are the same, exactly, as result with the use of the Hoffmeister-Polya technique.

The difference lies in the reconstruction of the continuity of the intestine. When it is feasible, gastroduodenostomy, or in other words completion of the operation in the Billroth I manner, seems better than, and preferable to, gastrojejunostomy. The former results eventually in greater comfort and better, more efficient, physiologic activity.

Wollaeger, Comfort, Weir, and Osterberg[1] studied a group of patients who had had partial gastrectomy with a full Polya anastomosis and found an excessive loss of fat and nitrogen in the feces, which caused a decrease in body weight unless a diet of sufficient caloric content was taken to compensate for the wasteful loss. These authors noted that patients who had mild or no digestive symptoms tended to lose less fat in the feces than did the patients who had severe digestive symptoms. Some of the more important possible factors which they considered accountable for the excessive loss of fat and nitrogen in the stools of persons who had undergone partial gastrectomy with a Polya anastomosis were (1) rapid emptying of the stomach, (2) intestinal hurry, (3) diminished flow of pancreatic secretions or bile because of impairment of the mechanism of stimulation of such flow, and (4) imperfect mixing of food with digestive ferments because of the shunting of gastric contents away from the duodenum.

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Our clinical impression is that patients who have had a Hoffmeister-Polya anastomosis have less postprandial distress and less difficulty in gaining or maintaining body weight than patients who have had a full Polya anastomosis, and that patients who have had a Schoemaker-Billroth I anastomosis do best of all. In fact, patients in the last group do not seem to suffer from the so-called dumping syndrome to any appreciable degree.

Wollaeger and Comfort[2] are concluding a study of intake and excretion on a group of patients on whom the Hoffmeister-Polya anastomosis was used, and also on a group on whom a Schoemaker-Billroth I anastomosis was done. Although their work has not been fully studied and the data evaluated as yet, the results of their studies seem to support our clinical impression.

TECHNIQUE

The essentials of the technique of the Schoemaker-Billroth I operation concern the delimitation of the resection and the closure of part of the stomach in order to form a tube of stomach which can be used in the subsequent gastroduodenostomy.

On exploration of the lesion after the abdomen is opened, several factors must be noted to determine whether this technique can be used. This will depend, of course, on whether partial gastrectomy is indicated and the lesion is operable. These factors principally concern the duodenum, but unusual inflammatory fixation of the stomach, especially of the portion remaining after resection, may prevent use of this technique. Considerable narrowing or stenosis of the duodenum or considerable inflammatory reaction and fixation of the duodenum will prevent gastroduodenostomy. Also, a duodenum which has been the site of peptic ulceration may be shortened so much that a gastroduodenal anastomosis may jeopardize the common bile duct, since in some such instances the papilla of Vater is only 2 or 3 cm. from the pylorus. Occasionally, especially in obese patients, the uninvolved duodenum will lack sufficient mobility to permit gastroduodenostomy after partial gastrectomy for a gastric lesion.

Mobilization of the stomach, with appropriate resection of omentum, mesocolon, and so forth, when indicated, and with ligation of the various blood vessels is done in the usual manner. The duodenum is transected in any suitable and preferred manner, but, if gastroduodenostomy is planned, a rubber-covered intestinal clamp should be placed distal to the site of transection.

Whether a Schoemaker-Billroth I or a Hoffmeister-Polya operation is to be done, the stomach is treated in exactly the same manner. At the elected site for transection two curved clamps are placed parallel to each other on the stomach, from the greater curvature, roughly perpendicular to this edge of the stomach, and the viscus is cut between them.

From the tip of the proximal clamp to the lesser curvature a Furniss clamp (actually any type of clamp may be used) is applied obliquely toward the esophagogastric junction; the degree of obliquity varies with the amount of tissue on the lesser curvature which it is desired to remove. Another curved clamp is applied just distal to the Furniss clamp to prevent spilling and the tissue is removed (Fig. 1).

The edges of the stomach transfixed by the pin of the Furniss clamp are united by means of an overlapping hemostatic suture (Fig. 2, *a*). The pin is removed and the suture is returned to the starting point as an inverting suture. A row of interrupted silk serosal sutures is then used for further reinforcement.

A rubber-covered clamp is then placed just proximal to the remaining curved clamp, and the latter is cut free with its contained, crushed bit of gastric tissue.

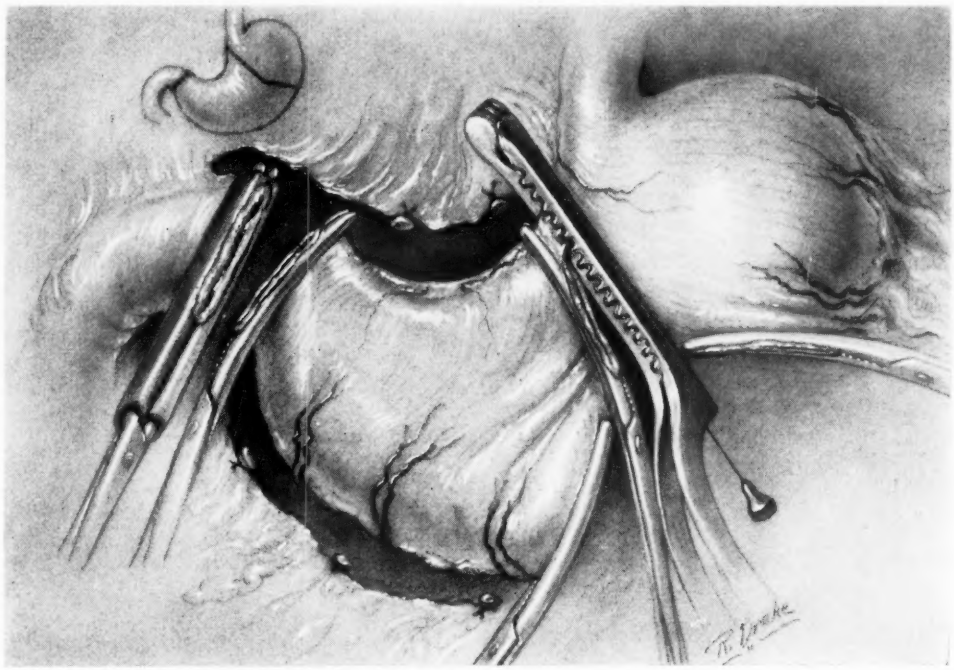

Fig. 1.—Rubber-covered clamp is placed across the duodenum just distal to the pylorus and the duodenum is transected. Two curved clamps are placed parallel to each other on the greater curvature and the stomach is cut between them to their tips. A Furniss clamp is placed obliquely across the stomach pointing up along the lesser curvature, another curved clamp is applied just distal to the Furniss clamp, and the stomach is removed.

The two rubber-covered clamps, the one holding the duodenum and the other the open end of the stomach, are then approximated for the anastomosis. Usually the lumen of the duodenum is smaller and must be fitted to the slightly larger gastric stoma. This bit of custom tailoring is achieved by means of a continuous posterior serosal silk suture, which begins and ends on the duodenum, and which, on being pulled tight, neatly approximates the two orifices (Fig. 2, *b* and *c*). The rest of the anastomosis is completed in a standard manner with two rows of catgut posteriorly and anteriorly, and interrupted silk serosal sutures anteriorly. The superior angle is reinforced by approximating the serosa of the superior aspect of the duodenum to the serosa of the posterior and anterior walls of the lesser curvature of the stomach with one or two silk sutures. To one of these latter is tied the superior end of the continuous posterior serosal silk suture, the inferior end being similarly tied and anchored. A bit of omentum is usually pulled up posteriorly and sutured over the superior aspect of the anastomosis for further protection.

RESULTS IN NINETY-FIVE CASES

Of the ninety-five patients in our group, seventy-three were men; their ages averaged 52 years and ranged from 30 to 77 years. Twenty-two patients were women; their ages averaged 51 years and ranged from 27 to 76 years.

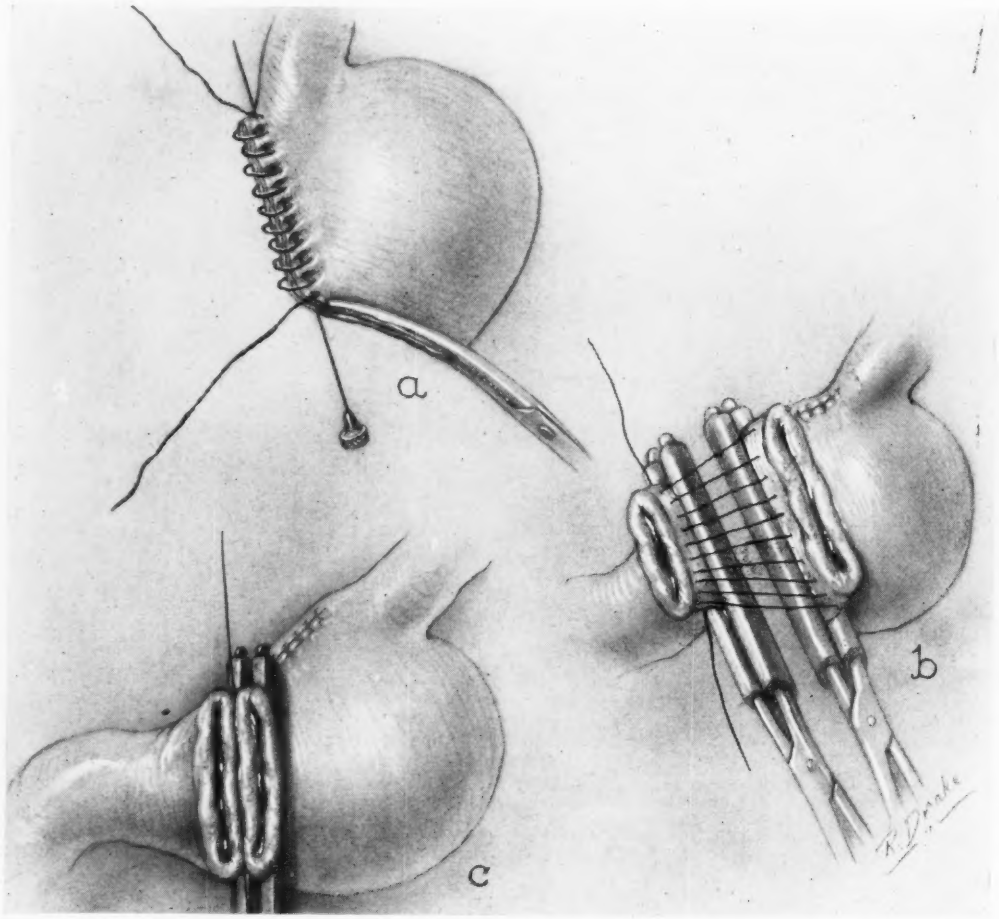

Fig. 2.—*a*, Lesser curvature portion of the end of the stomach is closed over the pin of the Furniss clamp. *b*, Rubber-covered clamp is placed across the stomach, and the duodenum and stomach are brought together. Continuous sutures are placed in the serosa of the duodenum and stomach; stitches are larger on the gastric side. *c*, Rubber-covered clamp is removed from the duodenum and the continuous suture is drawn up, approximating the two orifices and making them exactly the same size.

Results obtained for an elderly woman who had adenocarcinoma are indicated in Fig. 3, *a* and *b*.

The conditions for which gastric resection was done are listed in Table I. In only three cases in which duodenal ulcer was the primary lesion was it possible to perform a Schoemaker-Billroth I resection. Five patients had old or new duodenal ulcers accompanying more important gastric lesions (Table II). Accordingly, this type of anastomosis was possible in the presence of duodenal ulcer (of primary or secondary importance) in only eight instances.

This observation indicates, therefore, that the operation is most useful for gastric lesions. In so far as carcinoma of the stomach is concerned, it might be argued that proximity of the carcinoma to the pylorus contraindicates the use of a gastroduodenostomy, especially if there are enlarged nodes near the duodenum and head of the pancreas. It is our feeling that these factors are important only if resultant inflammatory reaction or carcinomatous extension has caused fixation and loss of mobility of the duodenum. As a matter of fact, such involvement might indicate inoperability in some instances.

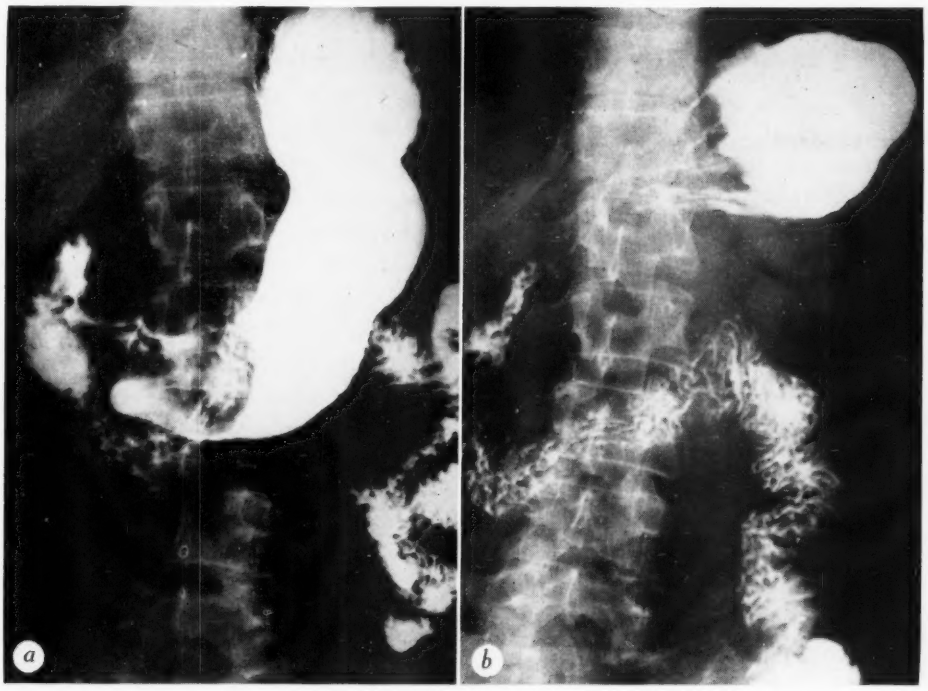

Fig. 3.—*a*, Preoperative appearance of the stomach of a woman 54 years old with an ulcerative adenocarcinoma, grade 4, who underwent operation on Oct. 16, 1945. *b*, Stomach of the same patient as visualized on Feb. 28, 1946. There is no roentgenologic evidence of recurrence.

In eighty-nine of the ninety-five cases, an estimation of the amount of stomach resected was recorded in numerical fractions. In sixty-four cases (72 per cent of the eighty-nine) the estimation was one-half, slightly more than one-half or three fifths. In all ninety-five cases the resected portion of the stomach was measured along a line midway between the two curvatures. In sixty-nine (72 per cent) of these specimens the measurement was 12 to 15 cm.

These estimations and measurements must be considered in the light of differences in size of various stomachs, and, furthermore, they are not truly indicative of the extent of resection of the lesser curvature. The Schoemaker technique with Billroth I anastomosis or the Hoffmeister technique with Polya anastomosis will permit as much as complete resection of the lesser curvature, which feature is not revealed by the method of measurement employed.

In twenty cases (21 per cent) associated operations were performed (Table III).

TABLE I. CONDITIONS FOR WHICH GASTRIC RESECTION WAS DONE

	MEN	WOMEN	TOTAL PATIENTS	
Peptic ulcer				
Duodenal	2	1	3	40
Gastric	30	7	37	
Carcinoma of stomach	32	12	44	
Chronic gastritis	4	0	4	
Gastrojejunal ulcer	2	1	3	
Multiple polyposis of stomach	0	1	1	
Lymphosarcoma of stomach	1	0	1	
Adenocarcinoma, low-grade 1, in adenoma of stomach	1	0	1	
Inflammatory cyst of stomach from gall bladder	1	0	1	
Total	73	22	95	

Leakage at the site of anastomosis occurred in two cases, was probable in one more, and questionable in one. In the last case the leak was suspected because of the drainage around the Penrose drain, but this was so slight and transitory that the presence of a leak was questionable. The probable leak occurred in the case in which postoperative gastric retention lasted forty-eight days, and a right subhepatic abscess was drained three weeks after operation.

Of the two patients who had definite leaks, one died on the ninth postoperative day and at necropsy separation of the suture line was found with an old local abscess and recent general peritonitis. In the other case the familiar fistula and infected wound developed and endured for fourteen days before healing.

TABLE II. DUODENAL ULCERS

	MEN	WOMEN	TOTAL PATIENTS
Duodenal ulcer			
Primary lesion	2	1	3
Accompanying gastric ulcer	3	1	4
Accompanying chronic gastritis	1	0	1
Total	6	2	8

Two hospital deaths (2.1 per cent) occurred in the series. One was the patient whose case was just described, the other patient died of a postoperative cerebrovascular accident. Both of these patients had had carcinoma of the stomach.

Although gastric retention developed in some of these cases as it does after other types of anastomosis, the retention was seldom severe or long lasting, and was easily controlled in most instances. Of the ninety-five patients, sixteen

TABLE III. ASSOCIATED OPERATIONS

OPERATION	CASES
Appendectomy	4
Disconnection of gastroenterostomy and closure of opening of jejunum, 3 With splenectomy, 1	4
Splenectomy and partial pancreatectomy	3
Cholecystectomy	2
Repair of postoperative ventral hernia	2
Splenectomy and partial hepatectomy	1
Splenectomy and excision of gastric leiomyoma	1
Splenectomy	1
Resection of part of transverse colon and double-barrel colostomy	1
Repair of epigastric hernia	1

TABLE IV. POSTOPERATIVE GASTRIC RETENTION IN SIXTEEN CASES

DURATION IN DAYS	CASES	POSTOPERATIVE DAYS OF OCCURRENCE*	EMESIS AND ASPIRATION (C.C. IN 24 HR.)	ADDITIONAL OPERATIONS
1	3	11 11 10	1,300 350 950	None
2	4	2 and 3 3 and 4 4 and 5 7 and 9	525; 275 1,700; 950 400; 450 200; 525	None
3	4	2, 3, and 4 2, 3, and 9 3, 4, and 5 2, 3, and 7	200; 500; 500 600; 200; 300 750; 1,050; 100 500; 350; 600	None
7	1	First 7	Usually large	None
9	2	First 9	Usually large	1. Anterior gastroenterostomy and entero-enterostomy 3 weeks after resection in 1 case 2. Patient died ninth postoperative day; separation of suture line
12	1	First 6; 11-17	Usually large	None
48	1	8-56	Usually large	Drainage of small subhepatic abscess and jejunostomy 2 weeks postoperatively; anterior gastroenterostomy seven weeks postoperatively

*Day of surgery is considered first postoperative day.

(16.8 per cent) had significant postoperative gastric retention (Table IV). The vomiting of quantities of 100 c.c. or less during the first thirty-six or forty-eight hours after operation or aspiration of such amounts in this period was not considered significant retention, nor was a single emesis of similar quantity considered to indicate retention even if occurring in the second week after operation. Significant retention was considered present if the vomiting of more than 100 c.c. of material occurred on one day, or on several days, successive or not, even if gastric aspiration after vomiting yielded no additional material.

In five of the sixteen cases the retention was of more than three days' duration. In four of these five cases retention began immediately after operation and in one on the eighth day (counting the day of operation as the first day). In eleven of the sixteen cases retention lasted three days or less. Those of one day's duration occurred on the tenth or eleventh day, while the others occurred within the first week for the most part.

The other postoperative complications in this series of patients are listed in Table V.

TABLE V. OTHER POSTOPERATIVE COMPLICATIONS

	TOTAL CASES
Urinary complications Retention (infections in 3), 28 Infection without retention, 1	29
Hemorrhage (mild hematemesis)	2
Infected wounds*	11
Venous thrombosis of legs	1
Pulmonary embolus and infarction	2
Postoperative atelectasis	2
Postoperative pneumonitis	3

*Peritoneal infections mentioned in text are included.

COMMENT

The Billroth I types of gastric operation are the most logical in so far as reconstruction of continuity of the alimentary tract is concerned and, of these, the Schoemaker modification is one of the easiest to perform, permits wide resection when desired, and yields the most nearly natural gastroduodenal continuity and relationship.

The Billroth I type of gastric operation has been much neglected in favor of the Polya forms of the Billroth II type. This neglect is largely the result of operations for duodenal ulcer for which only a Billroth II type of anastomosis is possible in most instances. However, it also has resulted from the general use of the full Polya anastomosis in cases of gastric carcinoma as well as duodenal ulcer. In this procedure the "straight-across" transection and use of the full mouth for anastomosis make a gastrojejunostomy almost mandatory. Only occasionally can a von Haberer-Billroth I operation be done with such a gastric stump, and then only if the duodenum is unusually large and mobile.

It is naturally true, and obvious, that a Schoemaker-Billroth I operation cannot always be done because of a fixed or immobile duodenum, a fixed stomach, a stenosed or inflamed duodenum, or because resection along the greater curvature of the stomach is of necessity high. However, the various duodenal and gastric features must be evaluated and considered in each case. This is well illustrated in the extreme in those cases in which esophagoduodenostomy has been possible after total gastrectomy.

From a physiologic standpoint the narrow stoma of a Schoemaker-Billroth I or a Hoffmeister-Polya anastomosis seems better than the wide stoma of a full Polya anastomosis. The Schoemaker-Billroth I anastomosis seems better than the Hoffmeister-Polya. The small stoma and the reconstruction of normal continuity by gastroduodenostomy seem to return the duodenum and stomach as nearly as possible to normal physiologic activity.

It might be argued that the small stoma resulting from use of the Schoemaker-Billroth I or the Hoffmeister-Polya technique predisposes to a greater incidence of postoperative gastric retention than does the larger stoma of the posterior Polya operation. It is possible also that this is more marked after the Schoemaker-Billroth I operation than after the Hoffmeister-Polya.

If, however, clinical impressions as to comfort, maintenance of body weight, and degree of postprandial distress are confirmed by factual laboratory studies (as they seem to be so far), then the order of preference, when possible, would seem established for these methods of gastric resection.

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A SUCTION AND FEEDING TUBE FOR THE POSTOPERATIVE CARE OF GASTRIC RESECTIONS

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THE surgeon is confronted with two important problems concerning the immediate postoperative management of patients who have had gastric resections. The first problem is that of adequate intragastric drainage and decompression to prevent pressure at the line of anastomosis with the jejunum. The second problem is that of meeting the nutritional requirements of the patient until he can take adequate food by mouth.

A Levine tube, with a Wangensteen suction apparatus attached, will provide adequate drainage for these cases but the nutritional problem is still unsolved. Water, chloride, carbohydrate, and vitamins can be administered parenterally but protein intake is inadequate postoperatively despite advances in parenteral hydrolysates.

Utilizing the principle of the double-barreled Abbott-Rawson tube, I have devised a simplified suction-feeding tube which has proved to be clinically effective in the postoperative treatment of gastric resections. The resections were performed at the Lankenau Hospital for gastric and duodenal ulcers and carcinoma of the stomach and in no instance has the tube caused untoward results.

The device is a size 18 French double lumen tube of soft rubber, 52 inches in length.* The distal end terminates in a hard rubber tip, sealing a column of mercury within the tube for a distance of 3 inches from the tip. Just proximal to the sealed mercury column are perforations for feeding purposes. Thirteen inches proximal to the tip is another series of perforations for intragastric suction. The proximal end of the tube bears a double lumen metal connection, one lumen of which is marked "F" for feeding and the other lumen marked "Suction." Accordingly, the feeding lumen terminates in the perforations at the end of the tube and is completely separate from the suction lumen which terminates in perforations well proximal to the tip. The tube is so divided that the suction lumen is of larger diameter than the feeding lumen, allowing adequate drainage of blood and gastric secretions postoperatively. The feeding lumen is ample for liquid protein preparations in a proper dilution.

Preoperatively, the patient undergoing resection is prepared by draining and lavaging the stomach with tap water or a weak solution of bicarbonate of soda until the drainage return is clear. This can be performed the evening before operation with a Levine tube, allowing the tube to drain by gravity

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*This suction-feeding tube is manufactured by George P. Pilling & Son Co., Philadelphia, Pa.

until the morning of operation. At this time, the Levine tube is removed and the suction-feeding tube may then be passed through the nose and pharynx. The passage of the tube through the nose is easily effected without injury to the mucosa and is facilitated by using a lubricant such as mineral oil. There is no tendency for the tube to coil back into the mouth owing to the weight and resiliency of the tip provided by the sealed column of mercury. The tube is then passed down the esophagus until the 18 inch mark is reached, at which point the tip lies just within the stomach. The tube is then secured with tape at the nose to prevent further movement.

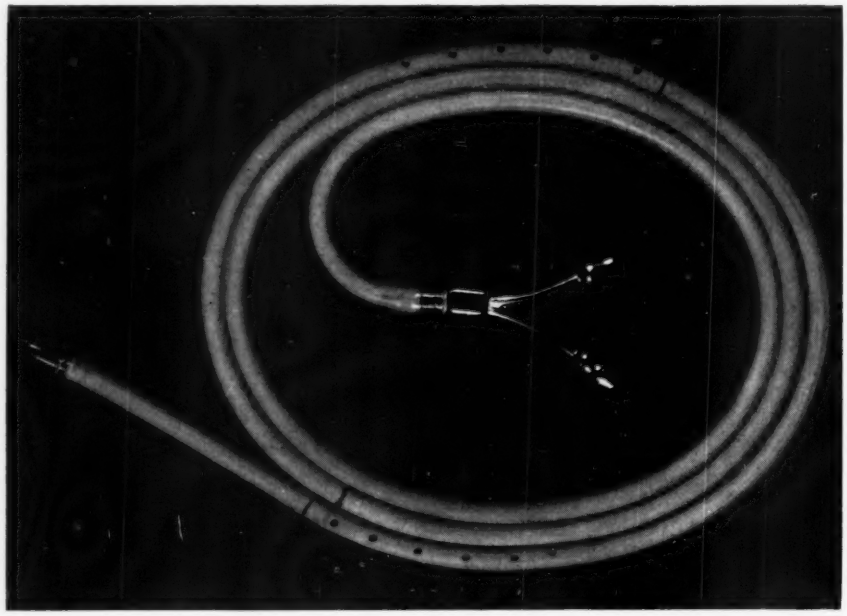

Fig. 1.—Size 18 French, double lumen tubing, 52 inches long. Terminal 3 inches of distal end contains mercury sealed with a hard rubber tip. Proximal to the mercury-filled tip are 6 holes, ½ inch apart, for feeding purposes. Thirteen inches from hard rubber tip are 6 holes, ½ inch apart, inserted in second lumen for suction. Tube terminates proximally in a metal connection, one lumen marked "F" for feeding and second lumen marked "suction."

The surgeon proceeds with the gastric resection and upon completing the posterior row of mucosal sutures of the anastomosis, the suction-feeding tube is freed at the nose and is advanced further by the anesthetist. The tip is then grasped by the surgeon, using a Babcock or Allis clamp, and is introduced into the efferent loop of jejunum. Continuing to advance the tube through the nose and guiding the tube through the anastomosis, the tip is advanced 13 inches into the jejunum. At this point the suction perforations appear and these should be allowed to lie just proximal to the line of anastomosis. The tube is secured again at the nose and the intubation is completed. Any looping of the tube which might occur as it is passed down the jejunum can be easily controlled by the assistant surgeon.

The anastomosis is then completed and the efferent loop of jejunum tends to fall away from the liver owing to the presence of the inlying tube. This

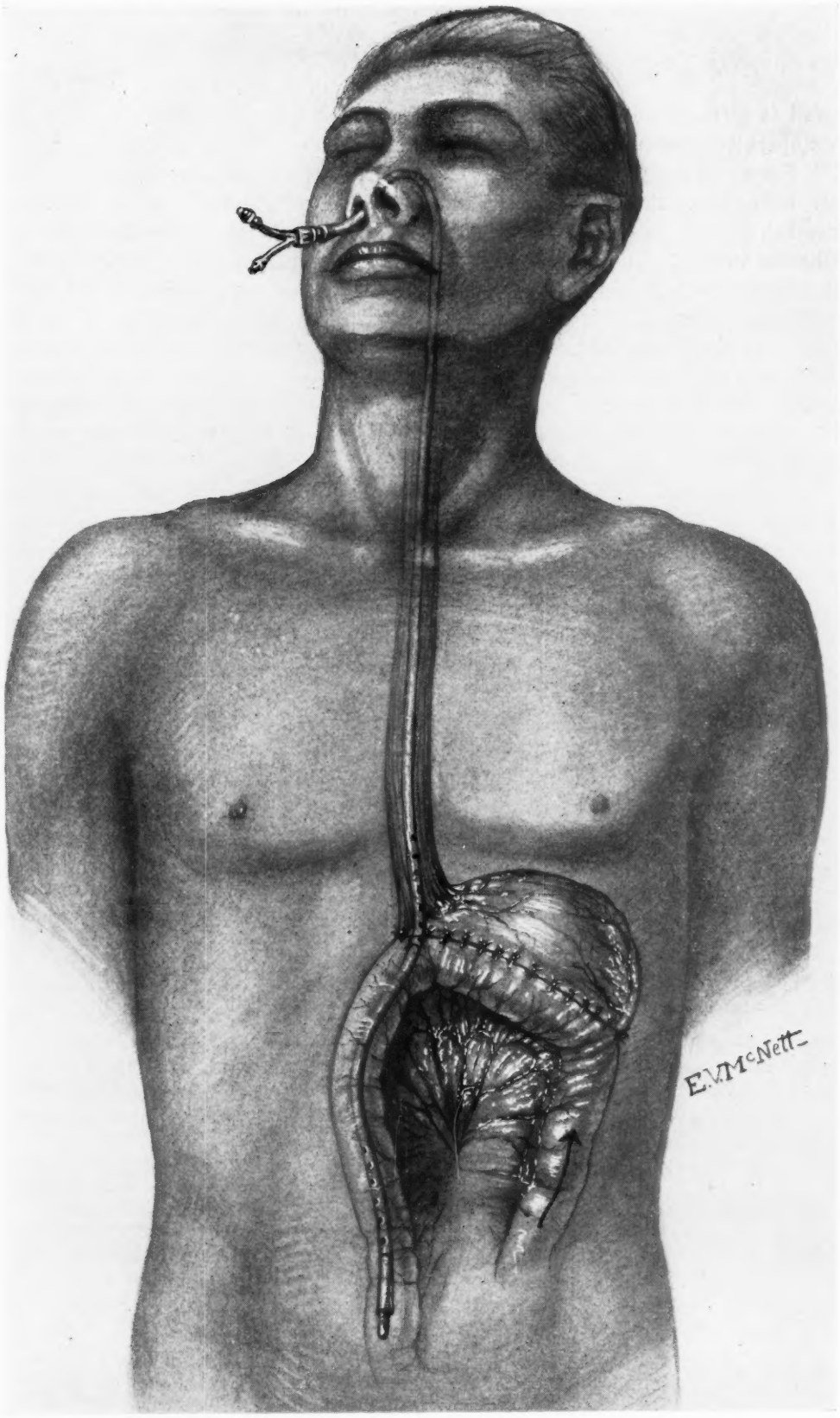

Fig. 2.—Diagram showing tube in place after operation. The distal perforations lie well down the fall-away, efferent loop of jejunum for alimentation purposes and the proximal perforations lie at the line of anastomosis for decompression and suction.

point is stressed because jejunohepatic adhesions are frequently the cause of postoperative obstruction following gastric resection.

Postoperatively, the double lumen metal fitting is equipped with short rubber connections and the suction lumen is handled precisely as an ordinary stomach suction tube, irrigating every two hours with tap water and applying constant suction. The feeding lumen is clamped until ten hours postoperatively at which time a liquid high protein digest, fortified with carbohydrate, iron, liver, and vitamins, is introduced by gravity flow. Initial feedings of 30 c.c. every two hours may be gradually increased every twelve to twenty-four hours, depending on the clinical picture. In a period of two or three days an optimum protein intake of 100 Gm. a day is achieved which can be checked by frequent serum protein determinations. An intake of 1,200 to 1,500 calories daily is also supplied by this type of feeding.

High protein, high caloric digests are prepared for tube feedings by the hospital dietitians and are made available in the quantity desired each day. Formulas for such aliments are available from various commercial houses.

This method of intestinal feeding, despite the usual postoperative ileus, provides nourishment by way of the most physiologic route and also provides a small bolus of food which may stimulate the bowel to regain its tone more rapidly.

Parenteral fluids are administered concomitantly to maintain fluid balance and caloric requirements. Oral liquid feedings in small amounts are usually instituted the first day postoperatively and gradually increased. When a positive balance exists between oral intake and suction drainage, the Wangensteen suction is discontinued and dependent on a positive balance with gravity drainage, the tube can finally be removed. This usually occurs about five days postoperatively. Once the suction-feeding tube is removed it is of no value to repass it and if distention occurs following its removal, a Levine tube should be used.

The suction-feeding tube can be used for gastroenterostomies as well as gastric resections; it causes no more discomfort than a Levine tube, it is cleaned and sterilized in the same manner as a Miller-Abbott tube, and the same tube may be used repeatedly.

SUMMARY

1. A simplified suction-feeding tube, devised for the care of postoperative gastric resections, has been found clinically effective.

2. A description of the tube, the method of intubation, and postoperative management is presented.

DIAPHRAGMATIC HERNIA FOLLOWING SUBDIAPHRAGMATIC VAGOTOMY

A Case Report

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THE present interest in section of the vagus nerves in the treatment of peptic ulcer and the increasing number of articles on the subject found in the literature may justify the report of an unusual complication, a diaphragmatic hernia following subdiaphragmatic vagotomy.

In a recent article Moses[6] reviewed the complications of vagotomy. The majority of the undesirable sequelae have been due either to incomplete section of the vagus nerves with failure to abolish the cephalic cause of gastric secretion, or to unpleasant physiologic side-effects accompanying the division of these nerves. The most frequently recorded of the latter are disturbances in gastric motility as evidenced by belching, fullness, loss of appetite, and vomiting.[4, 5, 7] Diarrhea is not infrequently experienced by patients following vagotomy. Dragstedt and associates[2] stated that prolonged gastric suction followed by slow progression of the dietpostoperatively will largely overcome the gastric atony and consequent symptoms. Our experience has tended to confirm these findings.

Achalasia was encountered by Moses[6] in one instance, relief being obtained by bromsalizol block of the celiac ganglion. Carlson[3] reported temporary stenosis of the esophagus at the level of resection of the vagus nerves, but a search through the literature failed to reveal diaphragmatic herniation as a complication of vagotomy.

A case of diaphragmatic herniation following vagotomy is summarized here.

CASE REPORT

C. P. (N. Y. H. No. 490067).—A white American married man, an instrument estimator, 37 years of age, was admitted to the surgical service of the New York Hospital Oct. 2, 1947, complaining of epigastric pain of three weeks' duration.

The patient had the onset of ulcer-type pain fifteen years previously and a duodenal ulcer was demonstrated by roentgen examination at that time. He was treated by dietary management and was asymptomatic for ten years.

Five years prior to this admission he began to have bouts of epigastric pain, relieved by milk and cream or alkali, recurring every six months and lasting about three weeks. Two years later he had tarry stools for three days during an exacerbation of pain. Three weeks before admission he had the onset of epigastric pain radiating through to the back, and nausea. The pain progressed in severity and he vomited six times during this period. No bloody or tarry stools, no jaundice, dark urine, or hematemesis were noted.

His past history was negative. Family history was significant only in that his mother had "a nervous stomach" and a maternal uncle died of duodenal ulcer.

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Physical examination showed blood pressure, 135/80; temperature 37° C.; pulse, 76; respirations, 20. The patient was well nourished and well developed, in no apparent distress, complaining of epigastric pain. Physical findings were limited to the abdomen where there was tenderness to palpation deep in the epigastrium.

Laboratory examination revealed urinalysis negative; blood: hemoglobin 15.5 Gm.; red blood cells 5.6 million; white blood cells 7.2 thousand; differential normal; Mazzini negative; blood urea nitrogen 13; blood sugar 104; serum protein 7.2 Gm.; three stool examinations showed guaiac negative; gastric analysis revealed a fasting free acid of 31 units, after histamine 53, 59, and 56 units of free acid at ten-minute intervals; gastric analysis after 20 units of regular insulin showed a rise to 46 units of free acid; night secretion (8 P.M. to 8 A.M.) 425 c.c. with 25 units free acid.

Fig. 1.—Roentgenogram of stomach and lower esophagus following vagotomy, demonstrating paraesophageal hernia.

Roentgen examination of the gastrointestinal series revealed an active duodenal ulcer, the duodenal cap being defective with an ulcer crater at its base. Findings from the chest roentgenogram, gall bladder series, and barium enema were negative.

On Oct. 11, 1947, subdiaphragmatic vagotomy was performed through a left rectus incision under cyclopropane-curare anesthesia. An active duodenal ulcer without evidence of obstruction was found. The triangular ligament of the left lobe of the liver was divided, a short transverse incision made in the peritoneum over the esophageal hiatus, and the lower esophagus delivered. Two large trunks of the vagus nerve were found and approximately 4 cm. of the posterior vagus and 3 cm. of the left were removed. Several small fibers were divided. The esophagus was allowed to fall into place and the left lobe of the liver was replaced. The abdomen was closed in layers of silk.

The patient's postoperative course was uneventful. He was placed on gastric suction for six days following operation and then the diet was slowly advanced without event. The postoperative night secretion was 250 c.c. and there was no free acid following the injection of insulin.

A gastrointestinal x-ray examination ten days after operation revealed a pouch arising at the esophagogastric junction and rising through the esophageal hiatus, the gastric rugae being traced into this pouch. The radiologist made the diagnosis of postoperative diaphragmatic hernia. The duodenal cap was still defective but there was no longer a crater (Fig. 1).

The patient revealed upon questioning that he occasionally regurgitated small amounts of food but that this was inconstant and caused him no trouble. He had had no return of the ulcer pain since operation and felt much improved. He was discharged on Oct. 30, 1947, and follow-up study was made in the outpatient department. On his first return visit two weeks after discharge the complaints were unchanged. He regurgitated occasional small amounts of undigested food, without apparent correlation to the consistency, but considered of no consequence.

DISCUSSION

The technique of vagotomy followed in this clinic is that described by Dragstedt and co-workers,[1, 2] who stated,[2] "When satisfied that the vagotomy is complete and that hemostasis has been secured, the operator closes the opening into the mediastinum with three or four catgut sutures." Prior to the case reported here, suture of the mediastinal opening had not been routinely performed by us, partly because it seemed unnecessary and partly because of the difficulty in exposing the esophageal hiatus in individuals with deep dimensions in the anteroposterior axis of the upper abdomen. Stimulated by this complication, reconsideration of the problem makes it seem advisable wherever possible to close the esophageal hiatus with silk sutures. It is readily understood how paraesophageal herniation may occur by relaxation of the hiatus due to spreading of the longitudinal fibers of the diaphragm in the delivery of the lower esophagus. The closure may be accomplished without difficulty in the majority of the cases.

SUMMARY

A case of subdiaphragmatic vagotomy followed by a hiatus hernia of the paraesophageal type is presented.

ADDENDUM

Since submission of this report for publication, the patient has been admitted and transthoracic repair of the hernia performed on May 28, 1948. When seen one month later he had no symptoms referable to either diaphragmatic hernia or ulcer.

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THE ROLE OF PLEURAL EXUDATION IN INFECTION FOLLOWING PNEUMONECTOMY

An Experimental Study

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ALTHOUGH there are no important structural differences between the pleura and the peritoneum, the pleura is usually regarded as having ineffective natural resistance to bacterial invasion. The rapid advances of abdominal surgery since the time of Lister developed an early understanding of the normal protective mechanism within the peritoneum. The use of the Fowler position was based on the mistaken belief that the pelvic peritoneum had greater bactericidal powers than that of the upper part of the abdomen. This was the beginning of the trend away from irrigation and free drainage of the contaminated peritoneal cavity which we now know can handle gross soiling under an airtight closure. Even today, there is no good evidence that any form of local chemotherapy will add to the natural powers of peritoneal protection except to afford a ready means of drug absorption into the circulation. Within the past decade, as the problems of anesthesia have been solved and our knowledge of thoracic physiology has advanced, intrathoracic surgery has become commonplace, but the ancient fear of pleural weakness is still widespread.

Much of this suspicion of the pleura may be traced directly to the writings of Robinson and Sauerbruch[1] and of Carrel,[2, 3] around the turn of the century. The prominence of these men gave weight to their opinions although neither offered experimental evidence to support his claims. Sauerbruch attempted to show that the pleural surfaces were much less resistant to bacteria than muscle, fat, or the other components of the thoracic wall but Noetzel[4, 5] had refuted such claims in his well-controlled but practically unnoticed work. Carrel had an enviable record in experimental vascular surgery in the thorax and maintained a very low rate of infection in his animals in a day when empyema was the usual sequel to thoracotomy. He protected the exposed pleura with oiled silk and practiced a meticulous technique which was no doubt the major factor in his successes. He maintained that the pleura was peculiarly susceptible to infection or trauma.

PREVIOUS STUDIES

A review of the many studies of pleural effusion produced by various irritants convinces one that the pleural surfaces react promptly and generously to inflammation. The misunderstanding and mishandling of this pleural response may account for the great variation in data regarding postoperative infections

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although Beaver and Mann[6] have pointed out that simple irrigation with hypotonic solutions is highly destructive to mesothelium. One must recognize that an open thoracotomy will expose most of the visceral and parietal pleura on one side, while even extensive laparotomy will subject only a fraction of the peritoneum to the drying effects of air. When partial or total pulmonary resection is done, a generous share of pleura is removed with the specimen and a large dead space remains to upset thoracic physiology and the spatial relationships of the thoracic viscera. Surgical dead space refers to an area which remains after surgical intervention, is isolated, enclosed by tissue, and contains only air and other gases. Enormous tumors may be removed from the abdomen without disturbing intra-abdominal pressures but the rigid thorax prevents rapid readjustment, which is afforded by the flaccid walls of the abdomen.

Clinical experience in World War II[7] has emphasized the importance of prompt removal of blood, fluid, and gas from the injured pleural space and rapid re-expansion of the lung, which is simply application of the ancient surgical principle that dead space must be eliminated. The modern operation of pneumonectomy[8] requires airtight closure of the thorax and simple readjustment of the intrathoracic subatmospheric pressure which favors full expansion of the remaining lung. Pleural effusion will collect and will eliminate the dead space; this effusion is rarely aspirated unless infection supervenes or an excessive collection is embarrassing the remaining lung.

The incidence of empyema following simple thoracotomy, operations on the heart, the great vessels, or the esophagus, or segmental pulmonary resection or lobectomy is surprisingly low compared to the appearance of this complication after pneumonectomy. Bronchiectasis, a lung abscess, or tuberculosis is each a common indication for pulmonary resection today and there is no doubt that a contaminated field is left in their wake.

Histophysiologic methods have been applied to the study of the surface membranes of both peritoneum and pleura for many years.[9, 10] Monroy[11] paid particular attention to the embryology of the elastic layers and Haas[12] and Latarjet and Francillon[13] investigated their relationship and distribution. Miller[14, 15] described the lymphatic relationships in the visceral pleura. Policard and Galy[16-19] studied the entire histologic structure of the various pleural regions in detail. Von Recklinghausen,[20] Maximow,[21] Kampmeier[22] and Mixter[23] selected the "milky-spots" for special study and Noel[24] reviewed and revised the conceptions of structure and function of these mesothelial layers.

Pleural fluid induced by various irritants has been widely investigated. Its cytologic content and reactions closely resemble those in peritoneal fluid. Borrel[25] showed a consistent sequence of polymorphonuclear leucocytes, followed by a mononuclear phagocytic infiltration in such exudates. Opie[26] pointed out that the polymorphonuclear leucocytes contained enzymes which were active only within definite limits of hydrogen-ion concentration, but Rous[27] concluded that the interior of the cell and its enzymatic activity were not influenced by hydrogen-ion concentration of the external medium. Kempner

and Peschel[28] demonstrated the tendency of an exudate to assume an acid reaction and Menkin[29] concluded that the cytologic picture in inflammation was dependent on the hydrogen-ion concentration of the exudate. In 1915 Lippmann and Plesch[30] caused the disappearance of granulocytes from the circulating blood of experimental animals with injections of thorium X and found that pleural exudates were then free of these polymorphonuclear cells, a fact which indicated the origin of these cells from the circulating blood.

Steinberg and Dietz[31, 32] showed that by a transfer of pleural exudates rich in polymorphonuclear leucocytes they could protect animals from an otherwise fatal pleuritis or peritonitis. However, supernatant fluid or cell suspensions did not give the same protection to animals as did whole pleural fluid. Linton[33] transferred stimulated omenta to the peritoneum of other animals, which did not then succumb to a usually fatal injection into the pleural space. He concluded that the recipient animals were protected by the mononuclear phagocytes of the transplanted omenta which migrated to the site of reaction in the thorax. Senn[34] had made similar observations on peritoneal reactions in 1888, and Thompson and Pollack[35] recently transplanted the omenta to the pleural space. Enhanced resistance was induced by Steinberg and Martin[36] in various species with graduated doses of heat-killed streptococci, which resulted in an increase of the mononuclear phagocytes in exudates produced later by bacterial irritants. Menkin and Warner[37] have shown by a series of experiments that pleural and other exudates contain a leucocytosis-promoting factor which is passively transmissible to other species.

Inflammation is almost universally regarded as a defense mechanism. The cytologic elements, be they polymorphonuclear or mononuclear, presumably offer the most inviting explanation for the *modus operandi*. Goodpasture and Anderson,[38] however, departed from these rather orthodox views, established by Metchnikoff,[39] and concluded that phagocytic cells may actually promote an infection by favoring growth and invasion by bacteria. Pokrovskaya and Makarov[40] concluded that the polymorphonuclear-clasmatocyte sequence formed a definite pattern for the excretion or absorption of an irritant. Robertson and van Sant[41] have found that both in vitro and in vivo the mononuclear cells of sterile effusions have greater phagocytic power than the polymorphonuclear cells in pneumococcic infections. Webb[42] has also performed passive transfer of phagocytes from rabbit to rabbit and thus was able to protect the host against otherwise fatal infections. Drinker[43] stated that regardless of cell type, abnormally large amounts of exudate will dilute the phagocytic cells and thus enhance the ability of bacteria to proliferate. In other words a cell-rich exudate will give greater protection against contamination than an exudate with fewer cells.

Wound healing, tissue repair, and disposition of bacteria, the criteria of "resistance," are doubtless related to nitrogen balance,[44] protein level,[45] vitamin deficiency,[46] and other factors, but they are more certainly dependent on the production of the cellular and humoral elements of the inflammatory response.[47] Cannon, Wissler, Woolridge, and Benditt[48] made the important contribution that in surgical infection prolonged hypoproteinemia, by virtue of depressed

gamma globulin fraction, could restrain antibody production. Many have shown that postoperative hypoproteinemia may be due to anesthesia, fasting, trauma, or loss of exudate and that it even accompanies minor surgical procedures.[49] It has not been suggested that a temporary depression of total serum protein may be the beneficent expression of the adequate inflammatory response as blood cells, antibodies, and serum, all rich in protein, are drawn into the involved region. Adams, Thornton, and Thomas[50, 51] have reviewed the causes of large loss of blood and of protein in thoracic operations and have attempted to prevent them by placing homogenous plasma in the pleural space after pulmonary resection in order to discourage the formation of the large amounts of exudates which invariably form at the expense of the patient's protein stores.

PURPOSE OF STUDY

The purpose of our study was manifold. We proposed to study the nature and origin of the pleural exudate which enters the pleural space after the removal of a lung, and to contrast it with exudates which have been produced artificially by foreign irritants. We wished to study the peripheral blood, during the time the exudate was entering the space, and to study the cytologic correlation which may exist between the blood and the pleural fluid. We planned to study the histologic changes in the pleural membranes during the time when the exudate was forming. The addition to the pleural space of various substances, such as plasma, drolan, or gelatin foam, in order to obliterate the so-called dead space, was studied.

Emphasis was given in this study to the immediate postoperative period, wherein infection was most likely to occur and when the exudate was rapidly accumulating. Observations have been made on animals up to one year after pneumonectomy but these data are not included.

METHODS AND PROCEDURES

Adult white rats, weighing 250 to 300 Gm. and free from any obvious disease, were used for this study. Healthy mongrel dogs were also used for comparison. The data assembled from our detailed study of the changes in the rat form the basis for this report although there were no important differences in the observations on these two species.

Prior to pneumonectomy a sample of heart blood was obtained from each rat. From this sample, the total erythrocyte and leucocyte counts, the hemoglobin level, the percentage of cells as determined by the hematocrit, the differential distribution of the leucocytes, and the total plasma protein level were determined. Standard techniques were employed in assembling the blood data and the Kingsley modified biuret method was used in the plasma protein determinations. Samples of pleural fluid were obtained from a series of normal rats under pentobarbital sodium anesthesia, using the abdominal approach and thence aspiration through the diaphragm. In this way sufficient amounts of fluid were obtained to assemble cytologic data with which the cytologic characteristics of the exudates could be compared. The mixture of blood from opening the thorax was avoided.

While the animals were under pentobarbital sodium-scopolamine anesthesia, and using a method of intratracheal insufflation previously described,[52] the left side of the thorax was opened and the entire left lung was surgically removed. Individual ligation of the pedicle structures was used in the dog but mass ligation was employed in the rat. Every attempt was made to prevent undue trauma to the parietal pleura, and drains were not used. In the rat it is possible to pull the pedicle out of the thoracic cavity for ligation and resection. In

so doing, blood was not liberated into the pleural space. Those animals which received the various solutions in the empty pleural space were injected with volumes of 0.25 c.c. per 100 Gm. of body weight, after closure of the thoracotomy incision. In order to favor adequate expansion of the remaining lung the intrathoracic pressure was adjusted to 3 to 5 cm. of water. When gelatin foam was used to replace the missing lung in the left pleural space, small portions of the substitute were moistened and used in amounts essentially equivalent to that of the removed lung. This material was packed into the pleural space after the costal sutures had been placed. The remaining lung was then fully expanded by increasing and sustaining the pressure in the endotracheal tube until the thorax had been tightly closed. The entire procedure, involving opening and closing the thorax, required about ten minutes. All animals in which any gross soiling of the pleural space had occurred during the operative procedure were not used in the computation of the data.

Animals were killed postoperatively in groups of three at intervals ranging from one hour to seven days. While the animals were under pentobarbital sodium anesthesia, the abdomen was opened and blood samples were obtained from the vena cava. Small samples of pleural exudate for cell study were obtained directly through the diaphragm, in the manner employed on the controls which had not undergone pneumonectomy. The thorax was then opened by removing the sternal plate. After the trachea had been ligated to prevent collapse of the right lung the amount of fluid present was noted and spatial relations of the heart and the right lung to the left pleural space were observed. We did not attempt to determine the exact amount of fluid present in all cases. This was difficult and the error in such determinations is great. Tissues were excised from the mediastinal, diaphragmatic, and parietal pleurae, fixed, and appropriately stained for histologic study.

RESULTS

1. Cytologic Characteristics of the Normal Pleural Fluid of the Rat.—Wide variations between species were noted in the total number of cells per cubic centimeter of fluid in the thorax of normal animals. The average of ten rats sampled was 46,700 cells per cubic centimeter, while that of dogs was but 4,500 cells per cubic centimeter. When smears of these fluids were studied differentially (Table I, control), the large mononuclear leucocytes, similar to those seen in peritoneal fluids and exudates and known variously as histiocytes, clasmatocytes, or macrophages, preponderated. These comprised in normal animals about 86 per cent of all cells. Small and large lymphocytes occurred in small numbers, and neutrophilic leucocytes were rare, but eosinophilic leucocytes represented about 9 per cent of all cells present. Occasional plaques of desquamated mesothelial cells appeared in most of the smears of the pleural fluids.

TABLE I. CYTOLOGIC CHARACTERISTICS OF PLEURAL EXUDATE AFTER PNEUMONECTOMY

TIME AFTER OPERATION	CELLS, THOUSANDS PER CUBIC MILLIMETER OF EXUDATE				
	LARGE MONONUCLEAR LEUCOCYTES	NEUTROPHILES	EOSINOPHILES	LYMPHOCYTES	BASOPHILES
Control	40.2 ± 6.5	0.2 ± 0.1	4.3 ± 0.7	0.4 ± 0.1	1.6 ± 0.2
1 hour	0.6 ± 0.1	6.0 ± 1.3	0.5 ± 0.1	5.3 ± 0.3	0
3 hours	0.2 ± 0.1	23.0 ± 2.6	0.1 ± 0.1	3.5 ± 0.8	0
6 hours	4.9 ± 0.1	22.9 ± 4.2	0.2 ± 0.1	3.1 ± 0.8	0
24 hours	18.6 ± 2.8	42.1 ± 7.7	7.6 ± 1.6	4.7 ± 0.3	0
72 hours	12.7 ± 1.1	14.6 ± 2.5	6.4 ± 1.1	4.5 ± 1.4	0
7 days	8.4 ± 0.1	1.2 ± 0.1	3.0 ± 0.8	0.1 ± 0.1	0

2. Cytologic Characteristics of Pleural Exudates After Pneumonectomy.—The changing character of the cellular constituents of the exudates, after pneumonectomy, is shown in Table I. One hour after operation, the total count had dropped from the accepted figure of 46,700 to 12,400 per cubic millimeter of fluid; of these approximately one-half (6,000) were neutrophilic granulocytes. Thus, within one hour after removal of the lung 5,800 neutrophilic granulocytes per cubic millimeter of pleural fluid had already entered the thorax. The number of these cells increased rapidly and at twenty-four hours after operation 42,100 neutrophilic granulocytes were tabulated for each cubic centimeter of fluid. As the volume of the exudate had obviously increased, this represented a tremendous collection of cells. The total number of mononuclear leucocytes per cubic millimeter of pleural fluid dropped very rapidly and was very low at one hour and at three hours after operation. The mononuclear leucocytes began to reappear in the exudate at six hours and were greatly increased at twenty-four hours after operation. At seven days, whereas the total count was low, the number of mononuclear leucocytes was now greatly in excess of the total number of neutrophilic granulocytes.

Small round lymphocytes, few in normal pleural fluid, were greatly increased in the exudates after pneumonectomy. These cells appear in the pleural space simultaneously with the fluid exudate containing granulocytes common to the blood stream and presumably they may be precursors of some of the large mononuclear cells which appear in large numbers later. The eosinophilic leucocytes, fairly abundant in normal fluid, were very scarce at three hours after pneumonectomy. They did not return in significant numbers until twenty-four hours after pneumonectomy. Their absence from the fluid in the early periods after operation, we believe, is due to their relative infrequency in the blood stream; their rapid infiltration later may be due to a proliferation and subsequent release from the bone marrow.

3. Blood Leucocyte Trend After Pneumonectomy.—The total lymphocyte and neutrophilic leucocyte counts made on rats at one, three, six, twenty-four, and seventy-two hours, and one week after pneumonectomy are shown in Table II. The total leucocyte count dropped 4,300 cells per cubic millimeter of blood at three hours after pneumonectomy. A return to the preoperative level was encountered at six hours but a second fall of 5,700 cells per cubic millimeter of blood occurred at twenty-four hours. At seventy-two hours, the

TABLE II. LEUCOCYTIC DATA OF BLOOD AFTER PNEUMONECTOMY

	LEUCOCYTES, THOUSANDS PER CUBIC MILLIMETER OF BLOOD			
	LYMPHOCYTES		NEUTROPHILIC LEUCOCYTES	
TIME AFTER OPERATION	BEFORE OPERATION	AFTER OPERATION	BEFORE OPERATION	AFTER OPERATION
1 hour	12.7 ± 1.2	9.6 ± 0.8	3.2 ± 0.5	5.9 ± 0.8
3 hours	10.8 ± 0.7	4.1 ± 0.5	4.3 ± 0.7	6.8 ± 0.7
6 hours	8.4 ± 0.1	3.6 ± 0.4	1.9 ± 0.3	6.9 ± 0.5
24 hours	11.8 ± 1.5	6.1 ± 0.7	2.9 ± 0.4	2.9 ± 0.5
72 hours	11.3 ± 1.3	9.6 ± 0.2	3.8 ± 0.4	6.9 ± 0.9
7 days	17.5 ± 0.9	8.8 ± 0.5	4.8 ± 0.7	4.0 ± 0.2

total number of leucocytes had returned again to its preoperative level. It will be noted (Table II) that the total number of lymphocytes dropped abruptly and remained below its preoperative level at each of the intervals recorded. The neutrophilic leucocytes, however, which were significantly above their preoperative levels at one and three hours, had reached a peak of 5,000 cells per cubic millimeter of blood above their preoperative levels at six hours. At twenty-four hours, the total number of neutrophiles was not

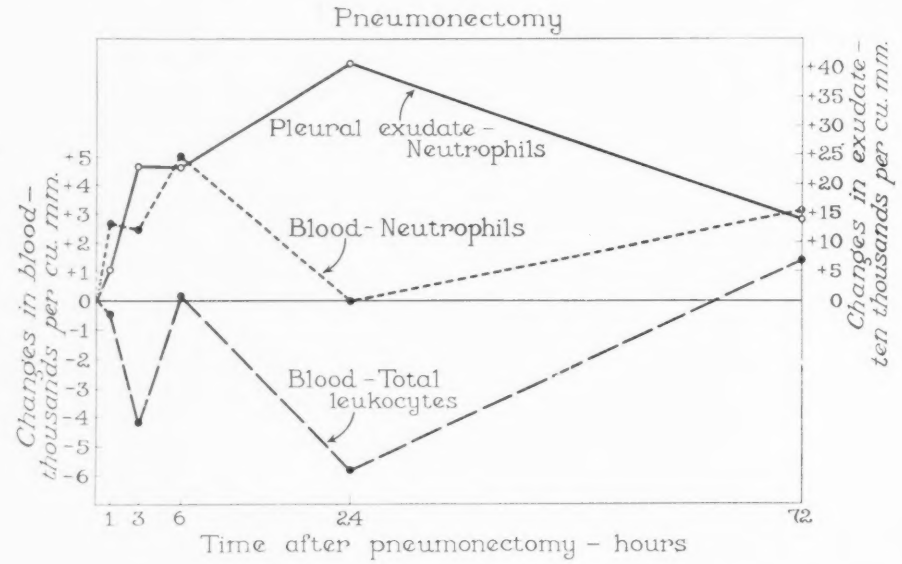

Fig. 1.—Changes of concentration of leucocytes in blood and exudate after pneumonectomy.

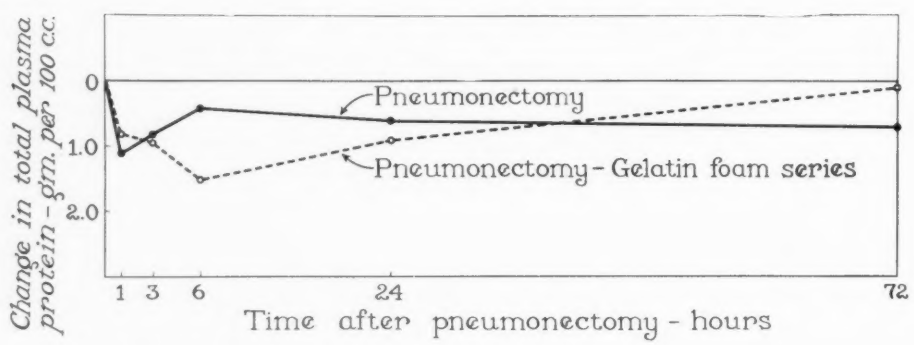

Fig. 2.—Changes in total plasma protein after pneumonectomy.

above the preoperative level but at seventy-two hours, when the postoperative total leucocyte count was again equal to the preoperative count, the number of neutrophiles had nearly doubled (Table II). The relationship of the total number of leucocytes and the number of neutrophilic granulocytes in the blood to the neutrophilic component of the pleural exudate at the various intervals is shown (Fig. 1). It will be noted that the neutrophiles of the pleural fluid reached their peak at twenty-four hours, at which time the neutrophiles of the

peripheral blood and the total number of leucocytes were both at their lowest levels.

4. Erythrocyte Count and Concentration of Hemoglobin After Pneumonectomy.—The data assembled on the erythrocyte counts and the hemoglobin levels, preoperatively, and at intervals varying from one hour to one week after pneumonectomy are shown (Table III). Since this operation entails only a slight loss of blood in the rat, it permitted a study of the effects of loss of lung tissue per se on these blood elements. It will be noted from the data assembled that there were no significant changes encountered, either in the total erythrocytic count or in the grams of hemoglobin per 100 c.c. of blood, in any of the pneumonectomized animals. Although declines in these two categories were apparent at seven days postoperatively, yet the statistical appraisal of the data indicates that the differences encountered cannot be regarded as significant.

TABLE III. ERYTHROCYTE COUNT AND CONCENTRATION OF HEMOGLOBIN AFTER PNEUMONECTOMY

TIME AFTER OPERATION	ERYTHROCYTES, MILLIONS PER CU. MM. OF BLOOD		HEMOGLOBIN, GM. PER 100 C.C. OF BLOOD	
	BEFORE OPERATION	AFTER OPERATION	BEFORE OPERATION	AFTER OPERATION
1 hour	9.28 ± 0.3	9.15 ± 0.2	14.1 ± 0.1	14.0 ± 0.5
3 hours	9.51 ± 0.1	8.43 ± 0.9	13.5 ± 0.2	13.0 ± 0.5
6 hours	9.28 ± 0.2	9.00 ± 0.2	13.7 ± 0.6	14.4 ± 0.6
24 hours	8.11 ± 0.2	8.90 ± 0.3	14.0 ± 0.6	14.1 ± 0.6
72 hours	9.63 ± 0.1	9.48 ± 0.3	15.7 ± 0.3	15.8 ± 0.9
7 days	9.92 ± 0.2	9.08 ± 0.3	15.3 ± 0.1	13.3 ± 0.7

5. Plasma Protein Levels After Pneumonectomy.—The total protein level in the blood plasma of fifty normal rats on which operation was not performed was found to range from 4.90 to 5.40 Gm. per 100 c.c. The average of the group was 5.25 Gm. and is represented by the zero line in Fig. 2. One hour after the removal of the left lung the level had dropped 1.1 Gm. from the control figure but at three hours the trend was upward and at six hours the average determination was only 0.3 Gm. below the accepted control level. Plasma protein levels did not return to the level obtained for normal animals during the postoperative seven-day period. This phase of the study simply prevented the inclusion of dehydrated animals and distortion of total cell count. The phenomenon of hypoproteinemia after major operations is well known.[53]

6. The Results of Placing Gelatin Foam in the Pleural Space After Pneumonectomy.*—The anatomic relationships in the thorax after removal of the left lung are shown (Fig. 3). The heart and mediastinum had shifted to the left; the ribs had retracted and the right lung had distended, with herniation of the postcaval lobe into the left thoracic space. The diaphragm and liver have been retracted somewhat to portray the dead space wherein the pleural effusion collects. When gelatin foam was placed in the space after removal of the lung, these changes in anatomic relationships were prevented (Fig. 4). Within seventy-two hours after operation, the gelatin foam had assumed the

*Gelatin foam supplied through courtesy of The Upjohn Company, Kalamazoo, Mich.

configuration of the missing lung. The mediastinal shift to the left was prevented, and the so-called dead space in the base of the left hemithorax had been largely obliterated. Effusion was present in animals packed with gelatin foam, but in amounts far less than those recovered from pneumonectomized animals without this material. In animals without the gelatin foam an average of 1.2 c.c. of effusion was recovered from the left pleural space twenty-four hours after pneumonectomy while in those packed loosely with gelatin foam an average of but 0.25 c.c. was recovered. This amount is only slightly in excess of that recovered from the pleural space of animals that did not undergo operation. It is obvious that such data cannot be exact but they do represent trends and indicate that less fluid accumulates when dead space is diminished.

Fig. 3.

Fig. 4.

Fig. 3.—Anatomic changes in thorax of rat eight days after left pneumonectomy.

Fig. 4.—Anatomic changes in thorax of rat seventy-two hours after pneumonectomy; the left hemithorax had been packed with gelatin foam.

The differential distribution of the cells of the exudate which developed in the thoracic cavity of the animals in which the dead space was packed with gelatin foam after pneumonectomy is shown in Table IV. A comparison of Table IV and Table I shows that, except at six hours, the total number of cells per cubic millimeter of fluid was less in the exudate of animals packed with gelatin foam than in that of animals which were not packed after pneumonectomy. In pneumonectomized animals receiving gelatin foam the largest number of cells (33,000 per cubic millimeter of fluid) was obtained in the exudate six hours after operation. In the series without packing, however, the total number of cells per cubic millimeter of fluid was greatest at twenty-four hours after operation when 73,000 per cubic millimeter were recorded for the exudate.

TABLE IV. CYTOLOGIC CHARACTERISTICS OF PLEURAL EXUDATE AFTER PNEUMONECTOMY IN GELATIN FOAM SERIES

TIME AFTER OPERATION	CELLS, THOUSANDS PER CUBIC MILLIMETER OF EXUDATE				
	LARGE MONONUCLEAR LEUCOCYTES	NEUTROPHILES	EOSINOPHILES	LYMPHOCYTES	BASOPHILES
Control	40.2 ± 6.5	0.2 ± 0.1	4.3 ± 0.7	0.4 ± 0.1	1.6 ± 0.2
1 hour	0.3 ± 0.1	0.4 ± 0.1	0.1 ± 0.1	1.6 ± 0.6	0
3 hours	1.3 ± 0.2	17.1 ± 0.2	0.8 ± 0.1	1.5 ± 0.4	0
6 hours	8.6 ± 1.0	23.9 ± 3.1	0.2 ± 0.1	0.3 ± 0.1	0
24 hours	7.8 ± 1.7	17.1 ± 2.2	0.9 ± 0.1	0.4 ± 0.1	0
72 hours	6.8 ± 1.7	6.4 ± 1.5	1.3 ± 0.1	1.6 ± 0.6	0
7 days	6.2 ± 1.0	2.4 ± 1.0	0.2 ± 0.1	0.4 ± 0.1	0

With the exception of the six-hour period, when the total number of cells was greatest in the gelatin foam series of animals, and of the seven-day period, the number of neutrophilic granulocytes was consistently lower at each interval than the number of these cells in animals which did not receive the gelatin foam. At one hour after operation there were 5,600 neutrophilic granulocytes per cubic millimeter of fluid less in the animals with gelatin foam than in those without it. At three hours after operation, there were 5,900, and at seventy-two hours, 8,200, fewer neutrophilic granulocytes per cubic millimeter of fluid in animals with gelatin foam than in those without it. At the end of one week, neutrophiles were more concentrated in the exudate of the animals with gelatin foam packing.

The data assembled on the leucocytes of the circulating blood of animals at the various intervals after pneumonectomy and gelatin foam packing are given in Table V. There were no significant changes in the total blood leucocyte counts at any of the postoperative periods recorded. The only significant stimulation occurred at six hours after operation when the total number of neutrophilic granulocytes had increased 6,500 cells per cubic millimeter of blood over their preoperative levels. The changes in the total number of leucocytes in the blood, the numbers of neutrophilic granulocytes in the blood, and the numbers of neutrophilic granulocytes in the pleural exudates of pneumonectomized animals with and without gelatin foam are shown in Figs. 1 and 5.

The data assembled from the tabulation of the erythrocytes and from the hemoglobin determinations of the pneumonectomized animals with gelatin

TABLE V. LEUCOCYTIC DATA OF THE BLOOD AFTER PNEUMONECTOMY IN GELATIN FOAM SERIES

TIME AFTER OPERATION	LEUCOCYTES, THOUSANDS PER CUBIC MILLIMETER OF BLOOD			
	LYMPHOCYTES		NEUTROPHILIC LEUCOCYTES	
	BEFORE OPERATION	AFTER OPERATION	BEFORE OPERATION	AFTER OPERATION
1 hour	14.1 ± 1.1	10.1 ± 1.2	2.4 ± 0.4	4.1 ± 0.8
3 hours	9.3 ± 0.7	5.3 ± 0.7	2.9 ± 0.2	5.4 ± 0.7
6 hours	10.0 ± 1.4	5.1 ± 0.8	3.6 ± 0.6	10.1 ± 1.8
24 hours	8.7 ± 0.5	6.5 ± 0.6	4.1 ± 0.3	5.4 ± 0.6
72 hours	12.3 ± 1.4	8.6 ± 0.6	2.4 ± 0.3	3.8 ± 0.8
7 days	12.1 ± 0.4	13.7 ± 1.7	3.9 ± 0.6	3.4 ± 0.2

foam are given in Table VI. It is obvious that there were no significant changes in the levels of erythrocytes during the seven-day postoperative period. A significant elevation above the preoperative control level in the hemoglobin concentration was recorded at the six-hour interval but since this increase was not observed at the later test periods, it presumably represents some artefact and is not accepted as true.

7. Influence of Various Intrapleural Drugs and Solutions.—Homogenous plasma, solution of acacia (6 per cent), gelatin, and drolan were placed in the pleural space of several series of animals which had undergone the same surgical procedure of left pneumonectomy. These materials were selected as blood substitutes which were in common clinical use and were employed in this study in an attempt to inhibit or to prevent the effusion and the protein depletion which were shown to occur after pneumonectomy. The principal result, however, was an increase of morbidity and mortality rates in each series of

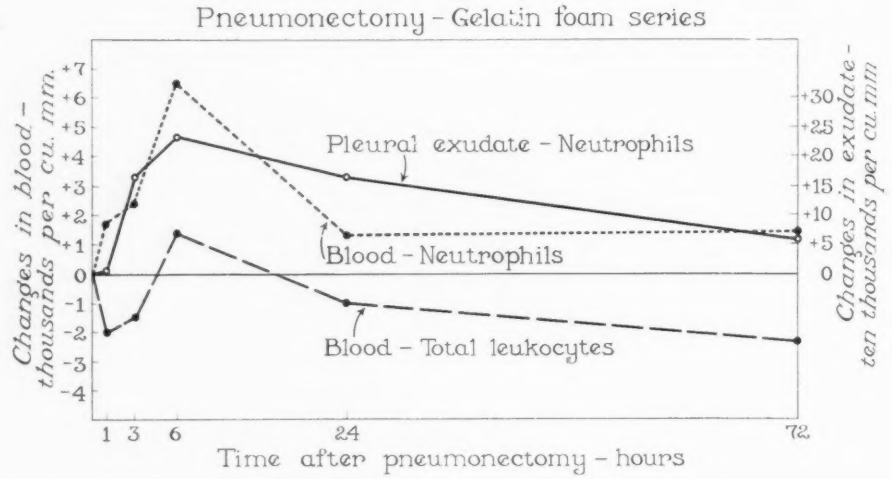

Fig. 5.—Changes of concentration of leucocytes in blood and exudate after pneumonectomy; the left hemithorax had been packed with gelatin foam.

animals so prepared. Hypoproteinemia was not prevented and the plasma protein levels followed the curve established for the pneumonectomized animals (Fig. 2). The total numbers of leucocytes which appeared in the pleural effusion were extremely inconstant and were at a low level for twenty-four hours. Eight of the group of thirty-two animals were dead within seventy-two hours as a result of empyema. Of the remaining twenty-four, six gave microscopic and gross evidence of infection when killed. Studies showed that these animals could tolerate approximately 0.25 c.c. per 100 Gm. of rat of a substitute fluid placed in the pleural space but amounts in excess of this caused either immediate and fatal collapse of the remaining lung or complete cardiac arrest.

In addition to the isotonic blood substitutes tested, similar amounts of an amino acid mixture (amigen), 5 per cent sulfathiazole solution, penicillin (5,000 units per cubic centimeter of isotonic saline solution), and streptomycin (3,000 units per cubic centimeter) were employed. In each of the eight animals

TABLE VI. ERYTHROCYTE COUNT AND CONCENTRATION OF HEMOGLOBIN AFTER PNEUMONECTOMY IN GELATIN FOAM SERIES

TIME AFTER OPERATION	ERYTHROCYTES, MILLIONS PER CU. MM. OF BLOOD		HEMOGLOBIN, GM. PER 100 C.C. OF BLOOD	
	BEFORE OPERATION	AFTER OPERATION	BEFORE OPERATION	AFTER OPERATION
1 hour	9.55 ± 0.4	8.51 ± 0.2	13.7 ± 0.5	13.5 ± 0.2
3 hours	8.24 ± 0.3	8.71 ± 0.3	12.7 ± 0.5	12.6 ± 0.4
6 hours	9.36 ± 0.2	9.90 ± 0.1	11.4 ± 0.4	15.4 ± 0.1
24 hours	8.90 ± 0.1	9.50 ± 0.2	14.5 ± 0.4	15.2 ± 0.9
72 hours	9.40 ± 0.3	9.20 ± 0.3	13.6 ± 0.4	13.4 ± 0.5
7 days	8.65 ± 0.3	8.50 ± 0.2	14.1 ± 0.4	13.6 ± 0.4

given the amino acid mixture, a massive reaction developed with effusion which was almost devoid of any cellular elements. Two animals died with empyema and two with a collapse of the opposite lung. The remaining four showed the characteristic hypoproteinemia without clinical ill effects. In three of nine animals treated with sulfathiazole solution, empyema developed and the animals died; the remaining six showed hypoproteinemia but survived. No deaths or complications resulted in each group of eight animals given streptomycin or penicillin in the concentration indicated. In comparison with these data a control series of animals which were pneumonectomized but not given substitution therapy all survived. Likewise those in which the dead space was eliminated by means of the gelatin foam packing all survived.

8. Reaction After Thoracotomy and Pneumothorax.—Ten animals underwent thoracotomy without pulmonary resection. These were killed at various periods thereafter. Air was permitted to remain on the side on which operation was performed, with partial collapse of the lung. In each instance there had developed a small pleural effusion exactly similar in cytologic character to that which appeared after pulmonary resection. In all cases there developed a mild hypoproteinemia which persisted for seventy-two hours but there were no significant changes in the leucocytes or the erythrocytes of the circulating blood.

9. Reaction of Pleural Membranes After Pneumonectomy.—Histologic study of the various pleural membranes revealed marked variations in the extent of the reactions. The first change observed was capillary dilatation in the submesothelial layers as early as twenty minutes after closure of the thorax. After one hour, when an effusion was invariably present, there was marked edema of the submesothelial layers, persistent capillary dilatation, and extrusion of erythrocytes and leucocytes through the pleural membranes. Mesothelial plaques separated from the pleural surfaces and there was noticeable swelling of the individual mesothelial cells. In the costal pleura the elastic layer, the endothoracic fascia, apparently acted as a limiting membrane, isolating the underlying intercostal muscles, for there were no changes in these deeper structures. In the diaphragmatic and mediastinal pleurae, these elastic layers were less developed so that tissue reactions involving the muscular layers of the diaphragm included edema and a separation of muscle fibers. At seventy-two hours after pneumonectomy, marked edema of the mesothelium of the mediastinal pleura

was present. This was particularly true in those regions containing fat or lymphatic tissues. Reactions within the pericardial membranes extended into the cardiac muscle where edema and a separation of muscle fibers had occurred. These changes were not observed in any of the pleural or pericardial membranes on the side of the remaining lung.

Fig. 6.

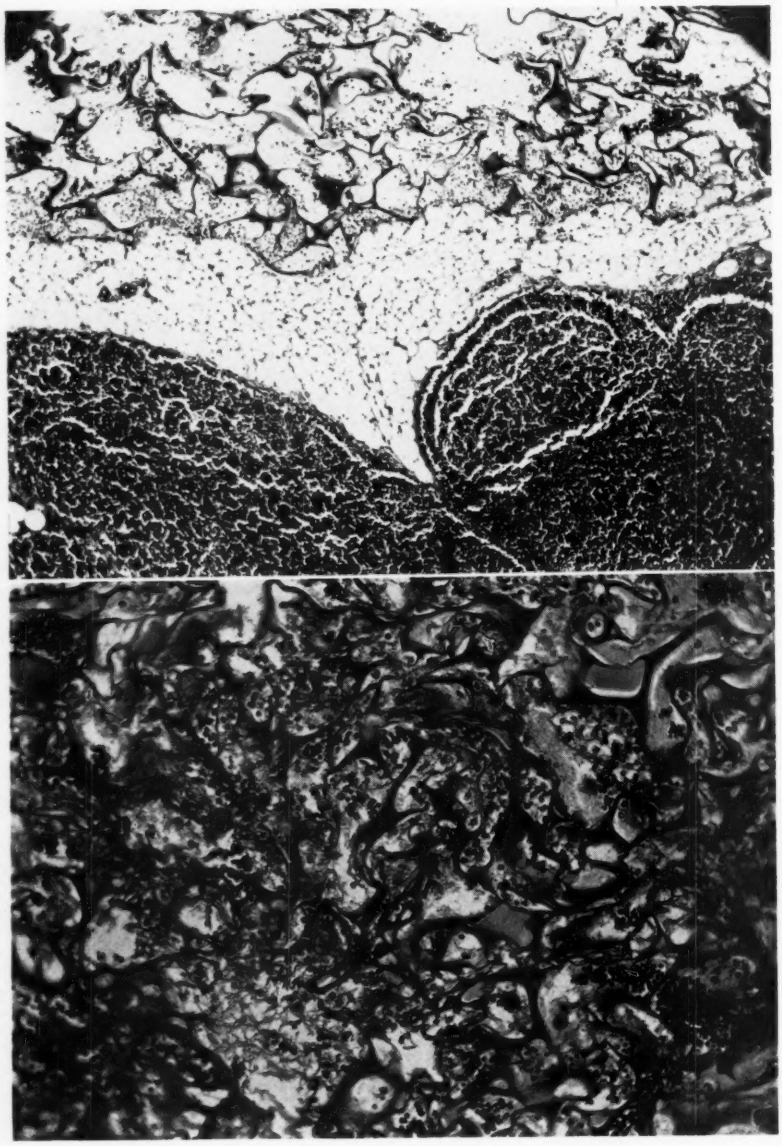

Fig. 7.

Fig. 6.—Gelatin foam packing attached to mediastinal pleura of rat, overlying lymph node, seventy-two hours after pneumonectomy (×50).

Fig. 7.—Gelatin foam packing from left hemithorax of rat, seventy-two hours after pneumonectomy (×125).

In the series of animals which had received the gelatin foam after pneumonectomy, marked cellular reactions developed in the mediastinal pleura where the gelatin mass had become intimately fused with it. Vascular capillaries extended from the pleura into the gelatin mass, and there was a marked proliferation and migration of fibroblasts into the gelatin. There were no cellular connections, however, between the gelatin mass and the parietocostal pleural membranes. The close connection of the gelatin mass to the mediastinal pleura of a rat seventy-two hours after operation in a region of adipose tissue which overlies a lymph node is shown in Fig. 6. A higher magnification (×125) of the same region (Fig. 7) shows the gelatin mass which apparently functions as a matrix for the extension of fibroblasts and capillaries from the mediastinal pleura. The cellular elements at the interface are largely mononuclear phagocytes and young fibroblasts.

COMMENT

Pulmonary resection, without surgical collapse of the thoracic wall as in thoracoplasty, produces a large surgical dead space, a condition which would not be encountered in the abdomen with its collapsible walls. The immediate and sustained reaction of the organism to pneumonectomy is obliteration of this space by a combination of factors which include (1) the overdistention of the remaining lung tissue, (2) the retraction of the parietes, and (3) the production of a fluid exudate. The later effects of pneumonectomy in lower animals cannot be compared to those in man since the patulous mediastinum in the former permits a gradual and complete expansion of the remaining lung which eventually fills the space. However, the early phenomenon of exudation is similar in both lower animals and man and appears presumably as a universal expression of irritation or a changed environment for the mammalian pleural membrane.

Packing the pleural cavity after removal of the lung with an inert nondistensible material such as gelatin foam serves to restore the remaining parietal pleura to a more nearly normal pressure status. Under such conditions the effusion which normally occurs after pneumonectomy is greatly inhibited but not completely suppressed and the cellular response in both pleural membranes as well as the exudate is but expressive of a mild inflammatory reaction. On the other hand, the cytologic change of the exudates and the histologic change of the pleural membranes which ensues on pneumonectomy without gelatin foam packing indicate an extremely high degree of irritation in the pleura. These pleural reactions appear to be comparable to changes which accompany bacterial or other forms of inflammation. The exudate produced by pneumonectomy was invariable in its time of appearance and the chemical, microscopic, and gross changes were always consistent and predictable. The closure of the thorax, followed by the regulation of the intrathoracic pressure to a constant level in pneumonectomized animals, has assured a set of regular conditions for the involved pleura, an adjustment which cannot be attained in clinical investigation owing to the varying ages and varying

pathologic states of the subjects. The pleural irritation which follows pneumonectomy appears to be on a purely mechanical basis and is not modified by buffering capacity of tissue, the pH, lymphatic blocking, or concentration of electrolytes. These are variables which need to be considered when various irritants are placed in the pleural space after operation.

The classic theory[54] for the origin of a pleural effusion is not applicable to the effusion which follows pneumonectomy, for these older studies eliminate the role of the parietal pleura and consider only the visceral pleura of dead animals in vitro. We were unable to find either gross or microscopic evidence that the visceral pleura of the remaining lung had taken part in the production of these pleural effusions. Graham's use of the concept of a changing negative pressure around the visceral pleura serves to emphasize the common misconception that such a negative pressure is in some manner essential to the performance of respiration, in which act it becomes still more negative. In senile emphysema or in the newborn mammal the lungs completely fill the pleural space, are almost lacking in elastic tissue, and do not collapse to any extent when the thorax is opened.

Small amounts of exudate were observed regularly when a simple pneumothorax was done and the cytologic picture of the resulting exudate resembled that found after pneumonectomy. The differences were only quantitative and were further expressed by the changes in total leucocyte level of the circulating blood. When the exudate was relatively large in amount, the large number of blood leucocytes which appeared in the exudate were coincident with a drop in total number of blood leucocytes. This relationship substantiates the earlier work of Lippmann and Plesch[30] who found no granulocytic cells in the exudates which formed from the irritated pleurae of animals whose circulating granulocytes had been destroyed with thorium X and indirectly shows the source of these cells from the circulating blood.

The granulocytic infiltration into the pleura thus represents the early phase of the pleural reaction to pneumonectomy. This phase is soon followed by the appearance in the exudate of large numbers of mononuclear leucocytes. This sequence of neutrophilic polymorphonuclear leucocytes, followed by an infiltration of mononuclear phagocytes, is the classic picture of the successful response to any inflammatory condition, be it bacterial or otherwise. These data do not sustain the views that either the microphage or the macrophage is the essential element in an inflammatory response but do establish the fact that the pleural response to injury is essentially similar to that observed in the reputedly more resistant peritoneum. The introduction of irritating substances into the pleural space has been shown to produce exudates which are large in volume but low in their cellular contents. The presence of inflammatory cells has been shown by passive transfer experiments to be essential for the destruction of bacteria and we have observed that exudates, low in cellular content, were invariably associated with an increased percentage of mortality and morbidity.

The hypoproteinemia which follows anesthesia and any surgical trauma was marked after pneumonectomy. No doubt this was partly due to the fact that the supply of circulating protein was tapped in order to provide for the

cells and serum of the exudate accumulating in the pleural space. Such hypoproteinemia has not been shown to be inconsistent with satisfactory healing in the otherwise healthy animal but it would seem rational to correct a too greatly prolonged hypoproteinemia in the debilitated human being. The placing of isotonic solutions such as plasma, certain blood substitutes, or various drugs in the pleural space after pneumonectomy did not obviate the lower plasma protein levels. Furthermore, these solutions invariably contributed to a dilution of the cellular elements which are required to combat contamination and to facilitate the repair process.

There was no depletion of the total number of erythrocytes or of the hemoglobin levels up to seventy-two hours after pneumonectomy, provided the animal was healthy and came to operation with a normal quota of these blood elements. At the later periods, however, we have observed significant declines in these measurements, which persisted for as long as six months.

Normal pleural fluid contains large numbers of cells which are recognized as the totipotent components of the so-called reticulo-endothelial system, the function of which is so largely related to reparative processes. This may presumably explain the known clinical and experimental facts which show conclusively that the intact pleural membranes without pneumothorax are capable of resisting heavy assaults. The high levels of leucocytes which exist within these normal fluids place them far from the conventional category of a mere thin film of lymph which acts as a lubricant.[55] If we are to regard the total leucocyte level in the circulating blood as an indication of the ability of the organism to combat or respond to an infection, then the enormous number of such cells within normal pleural fluid is certainly not without significance. Earlier studies[56] on the peritoneal fluid of albino rats have shown that a leucocyte level is maintained there which compares very well with the large number of such cells found in the pleural fluids. Furthermore, studies on the differential distributions have shown that the cellular components within the two fluids have almost an exact ratio to one another. The total leucocyte levels in the peritoneal and pleural fluids of the various species appear to be quite parallel to the known natural resistance which these common laboratory animals have attained. The rat is notoriously resistant to an infection of the thorax or abdomen and its almost complete resistance to the tubercle bacillus[57] is a most arresting fact when we compare the leucocyte count of its pleural fluid with that of the more susceptible dog.

The consistent character of the postpneumonectomic exudates, under the controlled conditions of the experimental procedures, was correlated with the histologic responses in the various pleural regions. The left diaphragmatic and the lower costal pleurae were found to be irritated in a manner similar to the parietal pleura, until such time as the effusion developed and apparently protected these structures from the irritating influences of the oscillating pressures incited by the respirations of the remaining lung.

The most marked and progressive changes were observed in the mediastinal pleura. In each animal in which the inert packing of gelatin foam had been inserted in the pleural space for more than forty-eight hours there was a

definite fibrinous attachment of the mass to the mediastinal pleura. This attachment was not observed on the costal or diaphragmatic surfaces until much later. At forty-eight hours, the fibrin content of the exudate, as seen in smears, had definitely increased, although quantitative estimations of fibrin were not attempted. The gelatin mass apparently acted as a matrix, for the capillaries and the fibroblasts extended into it from the adjacent mediastinum after eight days. Clotted fibrin was detected histologically in the interstices of the gelatin foam. A dense layer of clotted fibrin accumulated along the costal pleura forty-eight hours after pneumonectomy. Fibroblasts and capillaries extended eventually into the gelatin mass but this action was far less marked than from the mediastinum and the firm fibrous attachment of the gelatin to the costal pleura did not occur until much later.

Experimental observations indicate that the mediastinum is the site at which exudates largely appear and are removed from the pleural space. In this sense, therefore, the mediastinum serves the pleural space much as the omentum serves the peritoneal space.

CONCLUSIONS

The following conclusions have been reached in this study:

1. Small amounts of pleural fluid may be obtained from the pleural spaces of laboratory animals. Cytologically, this fluid is rich in mononuclear leucocytes and the concentration of these cells appears to bear some definite relationship to the natural resistance known to occur in certain species.

2. A pleural effusion invariably occurs after pneumonectomy. This effusion is the apparent result of an irritation of the diaphragmatic, mediastinal, and costal pleural membranes, incited by the changes in pressure exerted by the respiratory movements.

3. The cytologic patterns of these pleural exudates are definite and resemble those which obtain in any inflammatory reaction. The initial response is an infiltration of polymorphonuclear leucocytes in such tremendous numbers as to induce a simultaneous leucopenia and to indicate cell origin from blood stream reserves. The secondary response includes the infiltration of mononuclear phagocytes derived largely from adjacent tissues but partly also from the blood lymphocytes.

4. The dilution of effusions with either plasma, blood substitutes, or irritating drug solutions is accompanied by increased morbidity and mortality rates in these experimental animals. Moderate concentrations of penicillin and streptomycin in the pleural spaces do not disturb the cell content of exudates or unfavorably affect the mortality rate.

5. The amount of the pleural effusion after pneumonectomy may be greatly reduced by the elimination of the so-called dead space with an inert packing of gelatin foam.

6. The mediastinal pleura is comparable in certain functions to the greater omentum of the peritoneum.

7. An early postoperative decrease of erythrocytes and hemoglobin is due to loss of blood at operation and not to loss of pulmonary tissue per se.

8. The inflammatory response of the pleura is comparable to that of the peritoneum when this protective mechanism is not grossly disturbed.

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THE EFFECT OF STREPTOMYCIN, LOCAL AND SYSTEMIC, ON CONTAMINATED, SUTURED WOUNDS

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SINCE the time of Hippocrates surgeons have tried to reduce mortality and improve the healing of contaminated and infected wounds by the local application of a wide variety of substances. Recognition that most agents which kill bacteria also injure tissues finally led, in the 1920's and 1930's, to the virtual abandonment of wound antiseptics. Interest in local wound applications revived with the discovery of the sulfonamides. Being strongly bacteriostatic with slight, if any, toxic effect on tissues, much was expected of them; and at the beginning of World War II enthusiasm for the local use of sulfonamides was great. In carefully controlled studies of civilian wounds,[1] however, and in the extensive experience of military surgeons, sulfonamides applied locally were found to contribute little to the problem of wound infection. Near the end of the war their use was discontinued by the United States Army. The ineffectiveness of sulfonamides used locally is probably due to inhibition of their action by blood, pus, and tissue autolysates and to the relative resistance to their action of common wound contaminants, particularly the staphylococcus and gram-negative bacilli.[2]

The local use of penicillin in wound management has also been disappointing because of its narrow range of antibacterial activity, its transient effect, destruction of its activity by changes in pH, and inactivation by the products of many bacteria commonly found in wounds.[2] Although effective at times when repeatedly instilled in joint and pleural cavities containing susceptible organisms, local application to open and sutured wounds has been of doubtful value.

Streptomycin, with fewer limitations than the sulfonamides and penicillin, appears, in theory at least, to be a more suitable agent for application to wounds. It has a wide antibacterial range, being effective against both gram-positive and gram-negative organisms and against many organisms which are resistant to the sulfonamides and to penicillin. It acts rapidly in the presence of blood and pus and is relatively nontoxic to tissues. Streptomycin has been used in conjunction with Sulfamylon in the local treatment of experimental and clinical wounds by Howes,[3, 4] who believes that Sulfamylon may be more effective than streptomycin because of a wider antibacterial range and a greater resistance to changes in pH. There have been no reports, to our knowledge, of the local use of streptomycin alone under controlled conditions.

In the experiments herein reported, sutured wounds produced in a standardized manner have been inoculated with a standard dose of gram-positive and gram-negative organisms, a method resulting in 100 per cent wound infection

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and 53 per cent mortality in control animals. The efficacy of streptomycin in controlling the infection has been investigated by (1) systemic (intramuscular) administration of streptomycin after inoculation with the organisms, (2) local application of streptomycin at the time of inoculation with the organisms, and (3) local application of streptomycin eight hours after inoculation with the organisms.

EXPERIMENTAL METHOD

The Wound.—Wounds in dogs are notably resistant to infection, and a survey of the literature did not disclose a reliable experimental method of producing wound infection. The method of William White,* simulating traumatic wounds which become infected in human beings, has proved effective.

Operation.—The hair on the dog's back was clipped and shaved from the inferior angle of the scapula to just below the iliac crest, and the dog was anesthetized with intravenous sodium pentobarbital.† After preparation of the skin with iodine and alcohol, an incision 6.0 cm. long was made 2.0 cm. lateral to the spinous processes, beginning just below the last right rib. The muscle fascia was incised, and the incision was carried down into the underlying muscle to a depth of 1.0 cm. Silk ligatures were used for hemostasis. Six large Kocher clamps, 7½ inches in length, were applied to the muscle on one side of the wound, taking bites at least 0.5 cm. deep, and three of these were ligated with silk. The three remaining Kocher clamps were removed after two minutes. The same procedure was repeated on the muscle on the other side of the wound and on the subcutaneous tissue on both sides of the wound. One cubic centimeter of the broth culture of organisms was then introduced into the wound with a pipette, care being taken to prevent leakage, and the wound was closed. Five interrupted silk sutures were placed in the muscle and fascia and five vertical mattress sutures in the skin, all sutures being tied as tightly as possible without breaking the No. 00 silk. The wounds were covered with sterile gauze dressings secured with 2 inch adhesive strips wound completely around the dog's body.

The Organisms.—In order to produce wound infections similar to those in human beings, recent reports concerning the bacteriologic flora of infected wounds were studied.[5-11] The commonest wound pathogens appear to be the staphylococcus, streptococcus, and the colon bacillus, and these were chosen. Equal amounts of twenty-four broth cultures of *Staphylococcus aureus*, *Streptococcus hemolyticus*, and *Escherichia coli* were mixed in a test tube, and 1.0 c.c. of the mixture was transferred to each wound with a pipette, as described. Each cubic centimeter of the mixture contained approximately 45,000,000 staphylococci, 23,000,000 streptococci, and 425,000,000 colon bacilli. The streptomycin sensitivity of the organisms was *Staph. aureus*, 13.0 units per cubic centimeter, *Str. hemolyticus*, 0.33 unit per cubic centimeter, and *Esch. coli*, 1.0 unit per cubic centimeter.

*Formerly associated with the Harrison Department of Surgical Research. Unpublished data.

†Veterinary nembutal (pentobarbital Na), Abbott Laboratories, Chicago, Ill., each cubic centimeter of this preparation contains 1.0 gr. of nembutal; the dosage used was 0.4 c.c. per kilogram.

Streptomycin.—For local application to the wounds an ointment base was used as a vehicle, the composition of the base being carbowax (100 Gm.) and polyethylene glycol (125 c.c.).* Streptomycin was added to make a concentration of 5,000 units per cubic centimeter. Ten cubic centimeters of the ointment, containing 50,000 units of streptomycin, were applied to each wound with a spatula, special care being taken to prevent leakage of the broth culture and ointment.

In the animals treated systemically, 25,000 units of streptomycin per kilogram were injected intramuscularly every six hours for six days.

Experiments.—Four groups of animals were studied as follows.

Group A—Controls. The wounds were inoculated with the organisms and closed. No streptomycin was used (twenty-two animals).

Group B—The wounds were inoculated with the organisms and closed. No local streptomycin was used. Beginning two hours after the operation, streptomycin, 25,000 units per kilogram, was injected intramuscularly every six hours for six days (nine animals).

Group C—Streptomycin, 50,000 units in the ointment base, was applied to the inoculated wounds before closure (twenty animals).

Group D—The inoculated wounds were loosely closed to prevent leakage of the broth culture, and sterile dressings were applied. Eight hours later the wounds were reopened. Streptomycin, 50,000 units in the ointment base, was applied, and the wounds were closed with tight sutures, as in the other groups (ten animals).

The wounds were dressed and inspected every three days. On the sixth postoperative day blood cultures were taken, skin sutures were removed, and if there was any drainage, wound cultures were taken. Toxicity was noted.

Animals which died were autopsied. There was seldom any question whether death was due to anesthesia, distemper, or wound infection with septicemia. In case of doubt death was ascribed to wound infection.

RESULTS

The results of the experiments are summarized in Table I. Animals which died as the result of anesthesia or distemper are not included in this analysis. In Table II the blood cultures and wound cultures are tabulated.

In Group A, the controls, all of the wounds became infected and had positive cultures. In only two could the infection be considered mild, while in the others there were remarkably virulent undermining wound infections with extensive necrosis of the skin and subcutaneous tissue in most instances. In those which lived, complete healing did not occur for several weeks. Most of the animals were profoundly toxic, showing little interest in their surroundings or in food. Thirty-eight per cent had positive blood cultures. In five death was clearly due to anesthesia or to distemper, and in nine (53 per cent) of the remaining animals death was attributable to infection.

*The possible antibacterial effect of the ointment base per se was determined by the local application of the base without streptomycin to inoculated wounds in five dogs. The degree of infection and the clinical course in these animals were similar to the controls.

TABLE I. ANALYSIS OF EXPERIMENTAL DATA

CRITERIA OF LOCAL AND SYSTEMIC INFECTION	GROUP A CONTROLS (%)	GROUP B STREPTOMYCIN 25,000 U. PER KG. I.M. EVERY 6 HR. FOR 6 DAYS (%)	GROUP C STREPTOMYCIN 50,000 U. AT TIME OF OPERATION (%)	GROUP D STREPTOMYCIN 50,000 U. LOCALLY 8 HOURS AFTER INOCULATION (%)
Death due to local and invasive infection	53	0	14	0
Toxicity on 6th postoperative day	88	12	14	14
Purulent wound drainage on 6th postoperative day	100	37	14	28
Profuse	88	0	7	0
Slight	12	37	7	28
Wounds with extensive skin necrosis and severe undermining infection	65	0	7	14
Positive blood culture on 6th postoperative day	38	0	7	14
Positive wound culture on 6th postoperative day	100	37	14	28
Complete wound healing on 10th postoperative day	8	75	86	82

In Group B the prophylactic intramuscular administration of streptomycin resulted in a greatly altered postoperative course. In contrast to the toxic, lethargic animals in Group A (controls) most of these dogs were alert and active and ate well. There were no deaths due to infection and no serious infections. There were no positive blood cultures. In almost one-third there was slight purulent drainage when the sutures were removed on the sixth postoperative day, but signs of inflammation in these wounds were minimal. There was still

TABLE II. ANALYSIS OF BLOOD AND WOUND CULTURES

GROUP	BLOOD CULTURES POSITIVE (%)	BLOOD CULTURES STAPH. AUREUS (%)	BLOOD CULTURES STR. HEMOLYTICUS (%)	BLOOD CULTURES ESCH. COLI (%)	WOUND CULTURES POSITIVE (%)	WOUND CULTURES STAPH. AUREUS (%)	WOUND CULTURES STR. HEMOLYTICUS (%)	WOUND CULTURES ESCH. COLI (%)
A. Controls	38	86	50	33	100	93	75	53
B. Streptomycin, 25,000 u. per kg. I.M. every 6 hr. for 6 days	0	–	–	–	37	100	50	100
C. Streptomycin, 50,000 u. locally at time of operation	7	–	16	–	14	100	75	100
D. Streptomycin, 50,000 u. locally 8 hr. after inoculation	0	–	–	–	28	100	25	0

slight drainage on the tenth postoperative day, but healing was complete by the fourteenth day. Streptomycin blood levels in four of the animals six hours after an intramuscular injection of streptomycin were 4.0, 4.0, 3.0, and 2.0 units, respectively.

In Group C local application of 50,000 units of streptomycin in the ointment base at the time of inoculation with the organisms modified the infection to about the same degree as intramuscular administration in Group B. Most of the animals appeared unaffected by the organisms, and in all but two the wounds were healed on the tenth postoperative day. Two had positive blood cultures, and one, which died, had a severe wound infection with marked toxicity and skin necrosis. In several the wounds were fluctuant without signs of inflammation on the sixth postoperative day; and when the sutures were removed, the ointment was extruded. Coaptation of the wound surfaces following extrusion of the ointment resulted in rapid healing. In only two were the wound cultures positive.

In Group D local application of the streptomycin at a second operation, eight hours after inoculation of the wounds with the organisms, did not appear to be less effective than simultaneous application in Group C. The two groups are not large enough for statistical comparison. When the wounds were reopened, the tissues had lost their normal glistening appearance and were slightly edematous. One animal had a severe wound infection with a positive blood culture and died. Extrusion of the ointment when the sutures were removed was also noted in this group. In only two was there a positive wound culture, however, and all but two of the wounds were healed on the tenth postoperative day.

COMMENT

These experiments were designed to test the effect of streptomycin on contaminated, sutured wounds and to compare the effectiveness of local and systemic administration of streptomycin in preventing virulent wound infection. Although the number of animals studied is not large, streptomycin appeared effective in controlling both local and invasive infection.

A significant modification of the virulent infection of control animals by the prophylactic systemic administration of streptomycin was expected since the organisms were streptomycin-sensitive. A single local application of 50,000 units of streptomycin in an ointment base appeared to be as effective as intermittent intramuscular administration for six days. This was true both when the streptomycin was applied locally at the time of inoculation with the organisms and when applied eight hours later after multiplication and invasion of the bacteria had begun.

A single local application of streptomycin in these experiments apparently inhibited the streptomycin-sensitive organisms for a sufficient period of time to prevent serious infection in most instances, and this occurred in the presence of necrotic tissue. The different groups of animals studied are not large enough for statistical comparison.

The occurrence of streptomycin-resistant organisms in many human traumatic wounds would be expected to limit the clinical effectiveness of strepto-

mycin in the ointment base used in these experiments. In addition, the ointment base is not ideal since it causes separation of the wound edges and acts, to some extent, as a foreign body.

These experiments appear to support the concept that a single local application of an agent which is nontoxic, relatively stable, active in the presence of necrotic tissue, and has a sufficiently wide antibacterial range will aid in preventing infection in contaminated wounds. An ideal agent has not yet been discovered. Although superior to the sulfonamides and penicillin, streptomycin is probably limited chiefly by its incomplete antibacterial range and the lack of an entirely satisfactory method of keeping it in contact with bacteria within the wound for a long enough period of time.

CONCLUSIONS

1. In controlled in vivo experiments in dogs the effectiveness of streptomycin, systemic and local, in preventing mixed wound infections has been studied.

2. Using a standardized method of producing virulent, mixed infections in sutured wound in dogs, prophylactic systemic (intramuscular) administration of streptomycin was effective in controlling the infection, as evidenced by a reduction in mortality, toxicity, and septicemia (positive blood culture) and the elimination of severe wound infections.

3. A single local application of 50,000 units of streptomycin in an ointment base at the time of inoculation of the wound with organisms or eight hours later appeared to be as effective in controlling the infection as the systemic administration of streptomycin.

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A METHOD FOR PUNCTURING BLOOD VESSELS, LOCATED AND EXPOSED BY INCISION

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INFUSIONS or transfusions may become difficult and pressing problems when veins can be neither seen nor felt because of obesity or general circulatory collapse. A recently described method of infusing into the femoral vessels which uses the pulse of the femoral artery as a guide (Shaffer[1]) can be used only if this pulse can be felt; in an obese patient, or in a patient in circulatory collapse, this pulse often is impalpable. In such an emergency but two ways remain: bone marrow infusion or infusion into a vessel located by surgical exposure: "cutting down on the vein."

If surgical exposure is chosen, one may either transect the vessel and insert a cannula, rigid or semirigid, or an infusion catheter of soft rubber (Lamm[2]), or one may chose needle puncture. Either method has its advantages and disadvantages. Cannulation permits fluid administration over a comparatively long time, while needle administration usually is limited to hours. On the other hand, cannulation invariably leads to the eventual destruction of the vessel used, while needle administration usually preserves its patency. Cannulation is often technically difficult, while needle puncture should be easy, especially with the method to be described.

The principle of any good needle puncture of a vessel is that the angle between needle axis and vessel axis must be very acute.

An approach at a right angle between needle and vessel involves the following pitfalls: (1) The needle may push the vessel away rather than enter it. (2) If it does penetrate one wall of the vessel, it very easily may also penetrate the other wall. (3) The bevel may be longer than the inner diameter of the vessel and thus may lie only partly within its lumen. (4) Slightest movements of patient or operator may dislodge the needle. All of these dangers are reduced to a minimum if the needle approaches the vessel at a very acute angle.

If the incision exposing the vessel were parallel to it, the desirable "very acute angle approach" would be easy. Usually, however, one cannot use an incision parallel to the vessel, as one does not know its exact location, especially in just those cases where the need is most urgent, that is, when the patient is in collapse or is obese. In these patients, where veins can be neither seen nor felt, one will use an incision *transverse* to the course of the vessel (of which one has usually a good idea, although the vessel's exact location is not known). If one tries to puncture a vessel through such an incision, it will be approached at almost a right angle, especially if it lies deep, as in the obese patient. All the pitfalls enumerated here will be risked, that is, lateral displacement, transfixion, sticking-out of part of bevel, and easy dislodging.

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The advantage of the transverse incision, that is the ease with which it permits one to locate a vessel, can however be combined with the advantages of the "very acute angle approach."

A short segment of the vessel is exposed by transverse skin incision and blunt dissection of the subcutaneous fat. The infusion needle is then inserted *through the skin* a certain distance *away* from the incision (about three or four times the thickness of all tissue overlying the vessel), just over the presumed course of the vessel, such skin puncture being distal from the incision in puncturing a vein, and proximal when injecting into an artery. Then the needle point is advanced *subcutaneously* toward the exposed segment of the vessel, until it appears just over it in the depth of the wound. The vessel is punctured *under the guidance of the eye*, advancing the needle until all of its bevel rests well within the lumen.

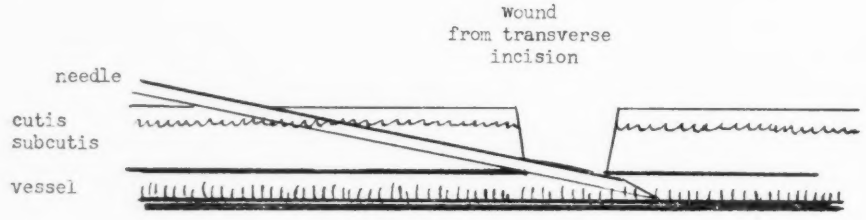

Fig. 1.

This method has been found useful in infusions and transfusions. It may also serve well in injecting into arteries, for example, in arteriography and in therapeutic arterial injections, as of penicillin solutions.

SUMMARY

Puncture of a vessel located and exposed by an incision transverse to its course can be done reliably and easily by approaching it with a needle which punctures the skin away from the incision and whose point travels subcutaneously until it appears in the depth of wound over the bared vessel where it punctures it at a very acute angle and under the guidance of the eye.

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SOME TECHNICAL CONSIDERATIONS IN THE ARTERIOGRAPHIC EXAMINATION OF THE LOWER EXTREMITY

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THE arteriographic examination of the lower extremity in various peripheral vascular states is an important addition to other tests now employed.[19] The method is not new, having been performed first upon an amputated extremity eleven weeks after the discovery of the roentgen ray.[3] Since that time it has undergone investigation intermittently[2, 8] but has never become a popular method of examination. This unpopularity has not been due to the paucity of information obtained, but rather to the cumbersome and difficult methods by which the procedure was accomplished. For this study a simple and uncomplicated method was devised. It is the purpose of this report to summarize the more important technical aspects of this procedure which have been gained in our series of over 100 arteriograms.

CONTRAST MEDIA

Since this method of examination was used for the first time in living subjects by Sicard and Forester, Berberich and Hiesch, and Barney and Brooks in 1923 and 1924,[2] many radiopaque agents have been utilized for intra-arterial injection.[1-3, 5] Most of these substances have fallen into disuse, however, because of undesirable side effects.[3, 12] Pain, for example, has been severe enough with some agents to require a general or spinal anesthesia.[2, 4] Others have necessitated surgical exposure of the artery to avoid extravascular deposition of the drug which otherwise would have resulted in extensive tissue destruction.[2, 4] Diodrast* and thorotrast[3] are among those which have survived a long period of trial. For our purpose Diodrast seemed to possess fewer disadvantages and was used in all cases in concentrations of 35, 50, or 70 per cent. Robb and Steinberg[10] and others[1, 13, 20] have studied extensively the occurrence of systemic reactions with the intravenous and intra-arterial administration of this substance. All precautionary measures advocated[16-18] to determine unusual sensitivity were done in our series, and no reactions were noted.

GENERAL CONSIDERATIONS IN THE SUCCESSFUL ACCOMPLISHMENT OF A SATISFACTORY ARTERIOGRAM

If this method of examination is to be superior to other tests now employed for the purpose of evaluating various types of peripheral vascular states, it must (1) reveal certain facts relative to the extent, type, and location of pathologic lesions, and (2) be easy to perform. The interpretation and classification of arteriograms are contained in another report.[19] In the performance of this method of examination, certain explanatory features should be enumerated.

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*Manufactured by Winthrop Chemical Co., Inc., New York, N. Y.

1. Primarily the object of the method is to visualize the functioning lumen of the arterial system of the lower extremity. Small collaterals entering the lower extremity cannot serve as sources of entry for the injection of radiopaque substances, thus, the first requirement is a patent major artery at a point where it enters the extremity. If this requirement is not present, the examination encompasses more than an extremity and as a result becomes more complicated.[15]

2. Arterial disease clinically present in the distal portion of the extremity frequently is a result of pathologic states in the proximal portion of the arterial tree.[6, 9, 19] For this reason, the radiopaque material must be injected at the most proximal part of the extremity and the entire arterial system be included in the examination.

3. The contrast media employed must be in sufficient concentration in the lumen of the artery to give good visualization on a roentgen film. Haszler[7] gave 5 per cent as the minimal intraluminal concentration of thorotrast for this purpose and found comparable results with iodine compounds. To obtain this concentration, it is important to use a needle of adequate size and equipment capable of rapid injection of the necessary calculated amount.

4. The mean velocity of the flow of blood in the normal femoral artery has been estimated to be 22 cm. per second.[21] Blood flowing at this rate through the artery will sweep the contrast media away before adequate visualization of the artery is obtained unless it is retarded.[3] In cases with occlusion of major branches, this rate will be considerably slowed, but to be sure that the velocity in all cases will be decreased it is necessary to occlude the femoral artery while injecting the opaque media until the film is exposed. This is best accomplished by digital compression of the femoral artery just above the site of injection.

5. The rate of diffusion of contrast media in arterial branches of equal size is markedly different if one is partially or completely occluded.[7] Thus, an open branch may have sufficient media for a good shadow long before the occluded branch. Such evidence recorded on a film could be interpreted as occlusion at the bifurcation. To avoid such inaccuracies, two complete roentgenograms with a short period of time interposed are required at each examination. The need for at least two serial films is further demonstrated in cases with segmental occlusion of major vessels. Blood (and contrast media) can only fill major vessels below such an occlusion by traversing smaller collaterals, which assist in slowing the rate of flow. In some cases, depending upon the extent of the segmental occlusion and the site of entrance of the filling collaterals, the flow in the major artery below the occlusion may actually be retrograde. Thus, the contrast media may be considerably delayed in reaching the distal portions of the leg.

6. To avoid pain at the site of injection and motion of the extremity during exposure, it is necessary to anesthetize the skin and arterial wall with procaine hydrochloride.[1, 3, 8]

7. The introduction of the needle through the skin and into the lumen of the femoral artery requires little explanation. No special ability is required, the

puncture being made under the direction of the palpating finger of the opposite hand, which is guided by the arterial pulsation.

8. If this method of examination were to be employed in many patients and conducted by a limited number of examiners, the question of overexposure should be considered. For the occasional examiner this would be of little significance. But if this factor was felt to constitute a real danger, the needle should be connected to the syringe by appropriate rubber tubing and the examiner should remain outside the field during exposure.

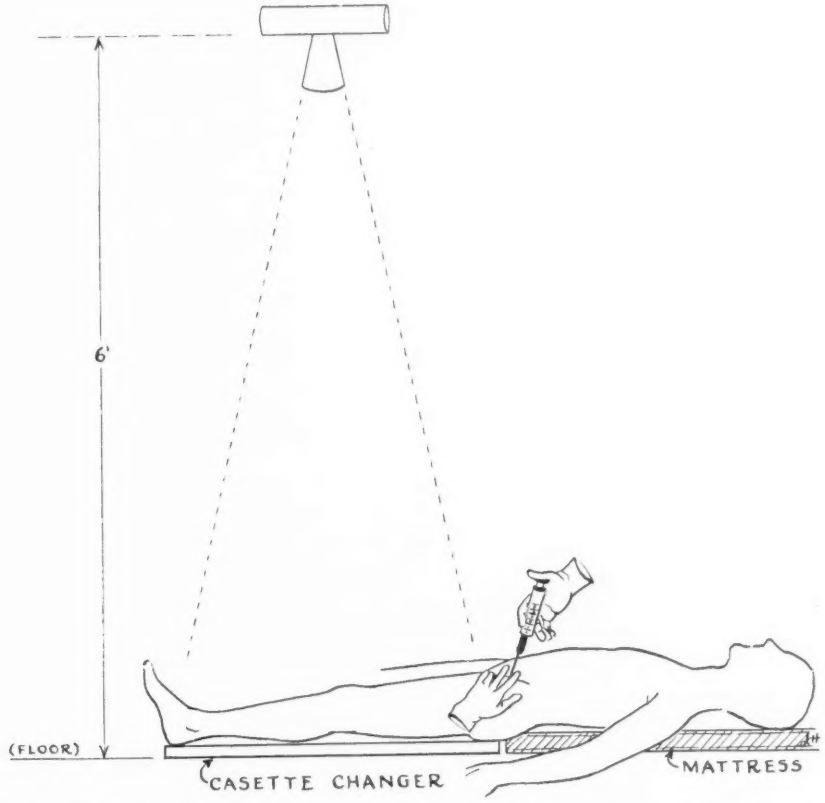

Fig. 1.—The patient in position for arteriography, lying on a mattress on the floor beneath the roentgen-ray tube. The involved extremity is in place on the specially designed cassette tunnel (Fig. 2). The position on the floor is necessary to obtain exposures at six feet with the ordinary radiographic equipment in which the tube cannot be raised six feet above the table. Exposures of this distance are needed to visualize the entire arterial tree of the lower extremity with one injection.

9. In order to change the cassettes as rapidly as desired and when desired without disturbing the position of the extremity, or the position of the needle in the artery, a cassette tunnel (Fig. 2) has been specially constructed. To expose the arterial system of the entire lower extremity, the patient is placed on a mattress on the floor at a distance of six feet from the tube (Fig. 1).

TECHNIQUE

The patient is placed on the mattress and cassette tunnel (Fig. 1) with the cassettes in the proper position in the tunnel (Fig. 2). The area below the

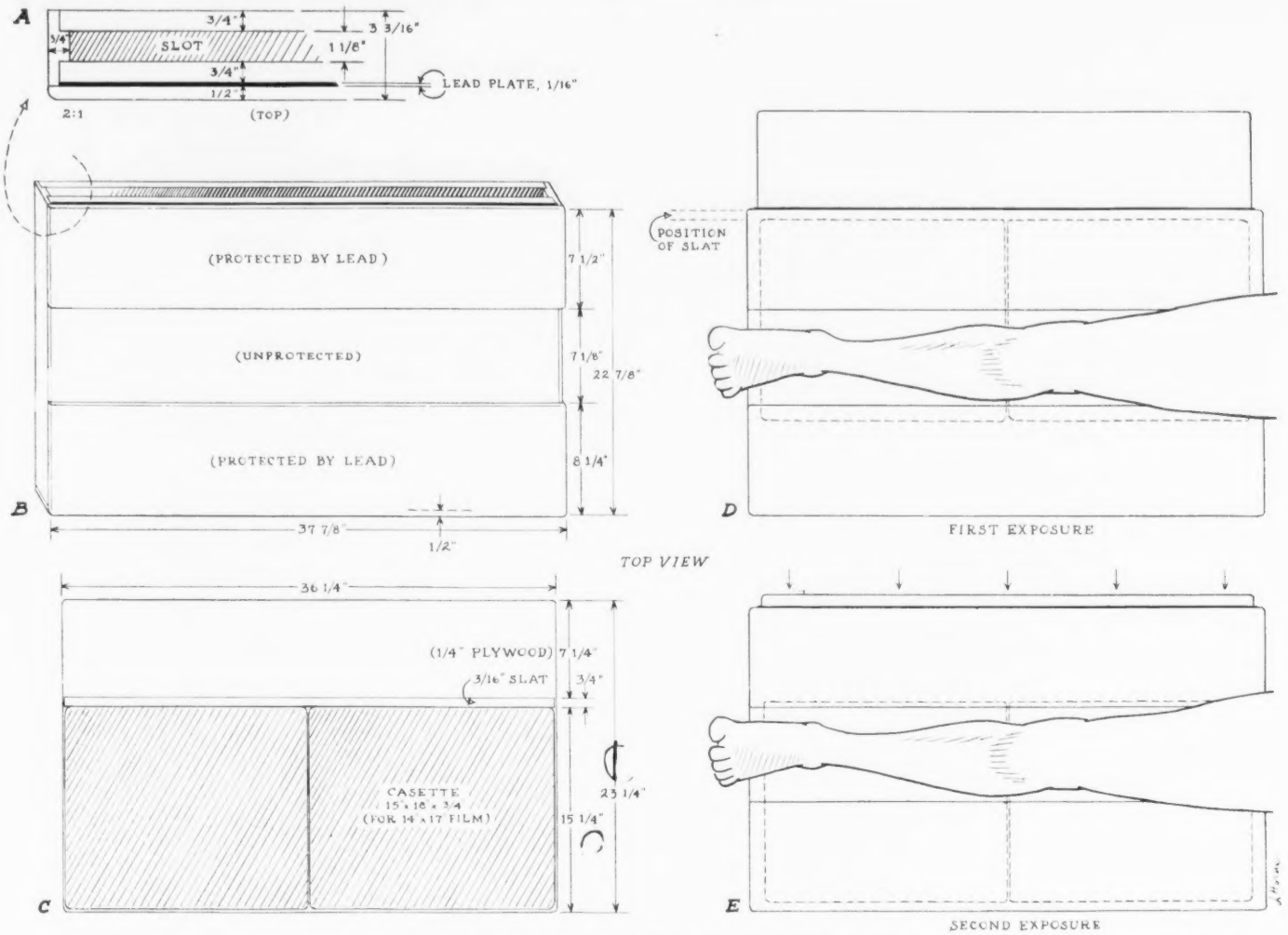

Fig. 2.—The cassette tunnel, or changer.* The central third is unprotected from roentgen-ray penetration, whereas each lateral third is protected by $^1/_{16}$ inch of lead (*B*). With the extremity placed over the center unprotected section of the tunnel the exposure occurs only on the portion of the film under this section (*D*). This is equal to one-half the width of the film being used (14 by 17 inches). By means of a slide which fits into the tunnel (*C*), the two cassettes are placed so that one-half of each film is protected by a layer of lead during the first exposure (*D*). For the second exposure the position of the film is changed by pushing the slide into the tunnel a distance equal to one-half the width of the film (*E*). This places the remaining unexposed half of the film in position under the extremity ready for the second exposure, and places the previously exposed portion of the film under the protection of the opposite sheet of lead. The final result is two roentgenograms of the arterial tree, side by side (Fig. 3).

*We acknowledge the assistance of Dr. Norman R. Shippey and Dr. Harry A. Miller in the design and construction of this equipment.

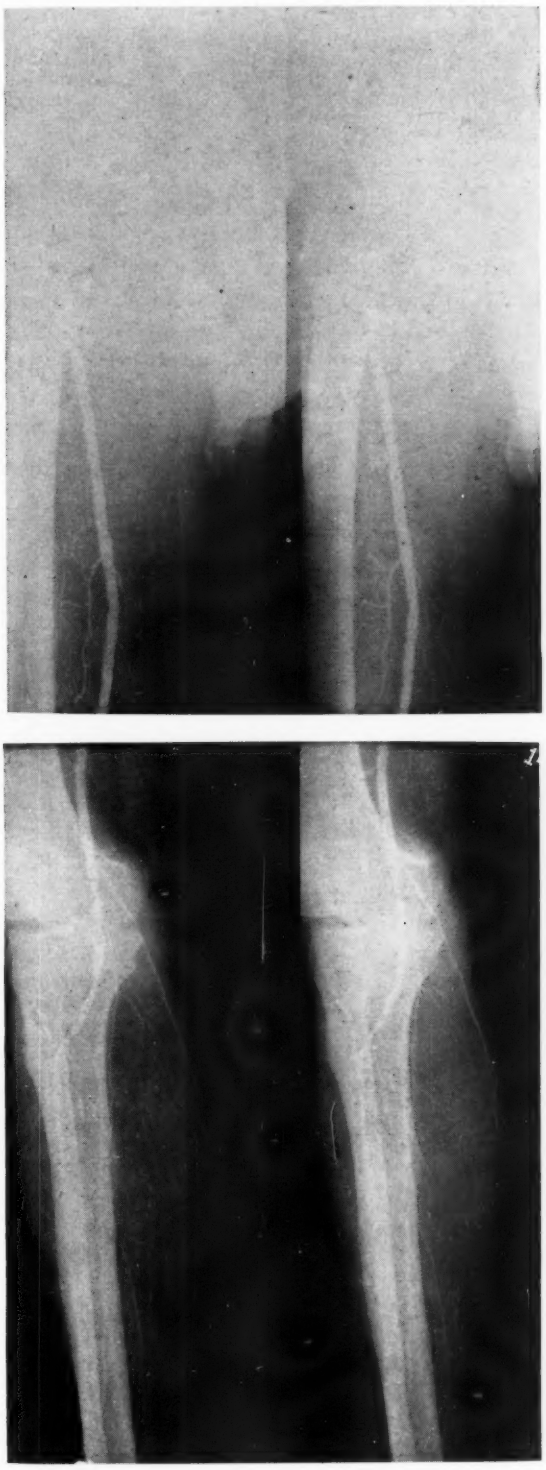

Fig. 3.—Roentgenogram of an arterial system obtained by the method described. There is 1¾ inch loss of visualization near the knee where the two cassettes are adjacent, the film not extending to the edge of the cassette.

*We acknowledge the assistance of Dr. Norman R. Shippey and Dr. Harry A. Miller in the design and construction of

inguinal ligament is prepared with suitable antiseptic and the artery palpated. The site of injection is anesthetized with 2 per cent procaine hydrochloride, infiltrating along both sides and above the artery. Thirty cubic centimeters of 35 per cent Diodrast are aspirated into a 30 c.c. Luer Lok syringe and this is connected to a long 18 gauge short-beveled needle. The needle with syringe attached is inserted into the artery under the direction of the palpating finger of the opposite hand. The fingers of the opposite hand are kept in position to occlude the artery above the site of injection, which is made about 1 cm. below the inguinal ligament. The artery is compressed against the pubic ramus and the Diodrast injected as rapidly as possible. When 25 c.c. have been injected, the first exposure is made. The tray of the cassette tunnel is pushed to position of second exposure (Fig. 2, *E*), and the digital pressure on the artery released for four seconds, meanwhile injecting the remaining 5 c.c. of the solution. At the completion of the injection the second exposure is made. The exposures are made at a distance of six feet, using 68 kv. and 300 Ma. for 1/20 second.

At the completion of the procedure the patient may become flushed and warm and should be forewarned.[10] Momentary pain in the leg during the injection has occurred in some patients.

SUMMARY

1. Some important features in the successful performance of arteriography of the lower extremity have been reviewed.

2. The technique of the method is described.

3. The details of the construction of a simple, inexpensive, but efficient cassette tunnel for the rapid changing of cassettes during the procedure is presented.

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THE VALUE DERIVED FROM UTILIZING THE COMPONENT PARTS OF THE TRANSVERSALIS FASCIA AND COOPER'S LIGAMENT IN THE REPAIR OF LARGE INDIRECT AND DIRECT INGUINAL HERNIA

A Group of Cases

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PRIOR to 1939 in all cases of repair of inguinal hernia I employed the Bassini operation or one of its modifications, in which the various deep layers of the inguinal space were sutured to the inguinal ligament. Recurrences resulted at a rate of 5 to 10 per cent in oblique hernia and a higher percentage in direct hernia. The majority of the recurrent cases were within the first year following operation. Seventy-five per cent recurred as direct hernias superior to the pubic tubercle and posterior to the external abdominal ring. The remainder recurred as indirect hernias, doubtless due to failure to remove the hernial sac or to inadequate closure of the internal abdominal ring.

Eight years ago in some cases of large indirect, direct, and recurrent hernias I began to utilize the component parts of the transversalis fascia, aponeurosis of the transversus abdominis muscle, and Cooper's ligament. For the last five years this type of operation has been employed exclusively in such cases. For the small hernia as seen in children, or in cases where the internal abdominal ring is not markedly dilated and the transversalis fascia is not impaired, the removal and closure of the hernial sac and closure of the ring are all that are required.

This study is based on 116 operations for large oblique, recurrent, and direct inguinal hernias. There were 98 patients, 18 of whom had bilateral hernias. A questionnaire was sent recently to each of the patients, asking if there had been any recurrence of hernia, and there were replies from all except 3, whose questionnaires were returned as unclaimed. In the answered questionnaires, without exception, the patients stated that there had been no recurrence. In only 2 cases was there infection following operation. After the infection cleared up there was no interference with or weakness of the inguinal support.

Ninety-one patients in the series were males and 7 females. There were 12 in the age group up to 19 years; 17 between 20 and 29 years; 16 between 30 and 39 years; 24 from 40 to 49 years; 14 between 50 and 59 years; 11 from 60 to 69 years; 2 in the group 70 to 79 years, and 2 patients in the ninth decade of life, both aged 81 years. The youngest patient was a boy of 12 years. The greatest percentage of cases (24.4 per cent) occurred in patients between the ages of 40 and 49, due to the fact, we believe, that this is the period of life when relatively strenuous activity is most likely to precipitate a break or giving way

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of the supportive inguinal tissues where there is a congenital weakness of the transversalis fascia.

Forty-nine cases involved the right side only; 31 the left side only; and 18, or 18.3 per cent, were bilateral. Seventy-four of the hernias, or 63.8 per cent, were oblique, and 42, or 36.2 per cent, were direct. There were four strangulated hernias. Five of the patients had had recurrences prior to this

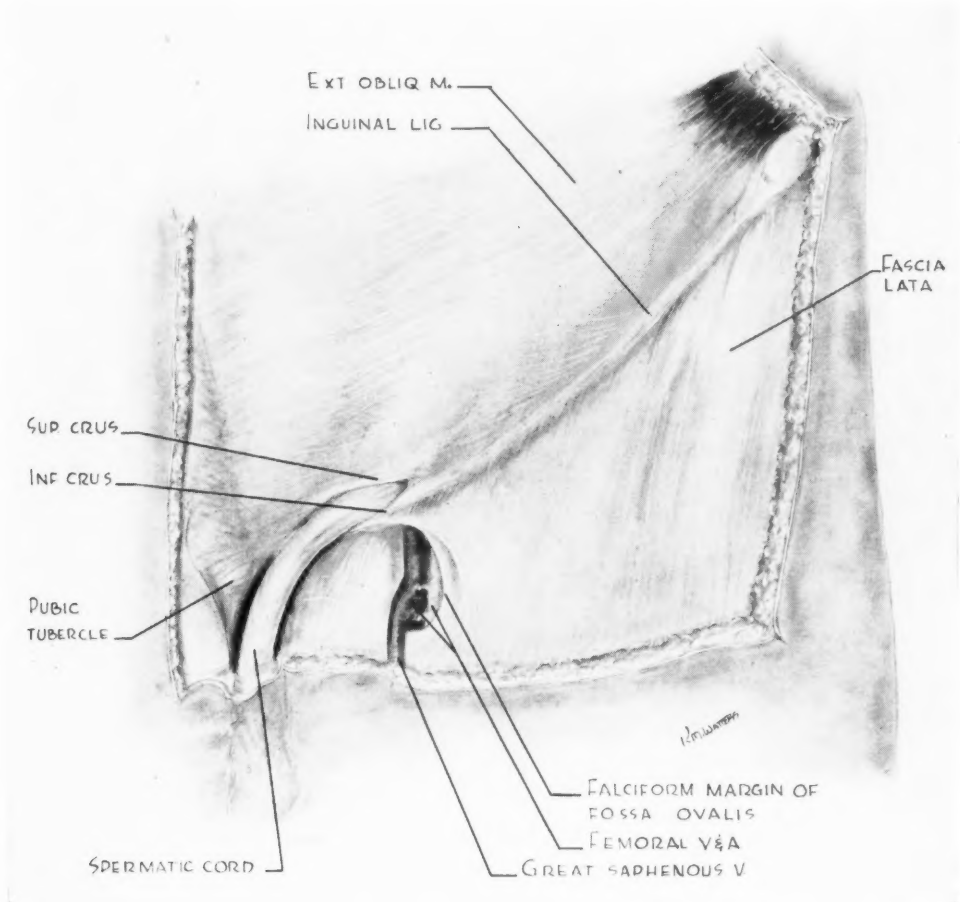

Fig. 1.—Superficial layer of the deep fascial structures of the inguinal region. Skin, superficial fascia, and superficial vessels have been removed.

time. The average size of the hernial cleft was 7.8 by 6.7 cm. The largest cleft was 13.2 by 10.1 cm. The average length of time in bed was ten to twelve days, as compared with eighteen to twenty-one days in the Bassini operation. The average length of time before return to work (hard labor) was four to six weeks. Lighter duties were allowed at the end of three weeks.

HISTORICAL

In recent years there has been a growing realization among surgeons of the need for fuller knowledge of the anatomy of the transversalis fascia, Cooper's

ligament, and the internal abdominal ring in the repair of hernia. This realization has been widely reflected in the literature.

The surgical utilization of the transversalis fascia and attached structures, including the transversus abdominis aponeurosis, together with Cooper's ligament, in hernial repair is, however, no longer new. In 1914 Testut and Jacob[14]

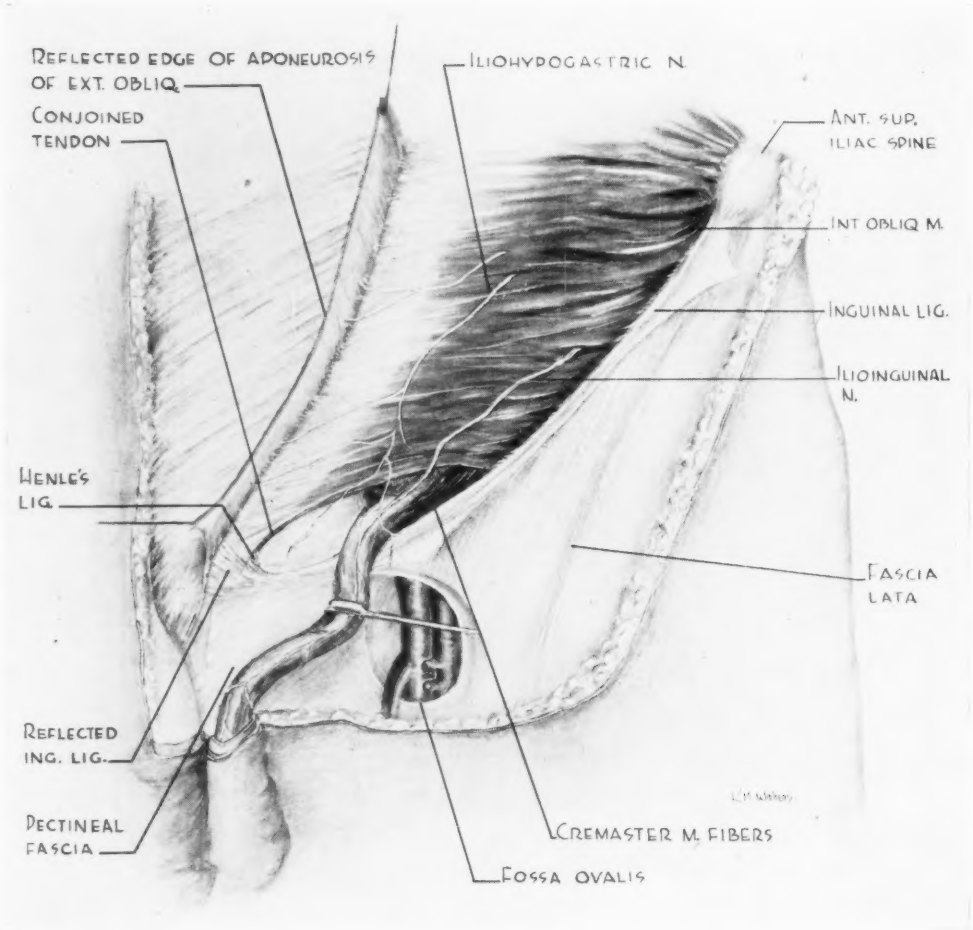

Fig. 2.—Intermediate musculofascial and nerve structures of the inguinal region.

described in detail the component parts of the transversalis fascia and Cooper's ligament. In 1938 Anson and McVay[2] found, after a study of 125 cadavers, that the aponeurotic portion of the transversus abdominis muscle showed a marked variation in its position in the inguinal region. In only 3 per cent did the muscular portion extend to the level of the spermatic cord. In 62 per cent it terminated in the superior half of the inguinal region, and in 7 per cent it was not inguinal at all. In approximately 19 per cent the layer was almost as aponeurotic as the external oblique in the same subject, the muscle fibers terminating at or near the interspinous line. Medially also there was considerable variation in the extent of the muscular part of the layer. In only about one-

half the cases did the muscle fibers form the lateral two-thirds of the stratum in the inguinohypogastric area and the aponeurosis the medial one-third, as is consistently the arrangement for the internal oblique. Subsequently (1942) McVay and Anson[11] pointed to the error in the Bassini operation of approximating the transversalis fascia to Boupart's ligament.

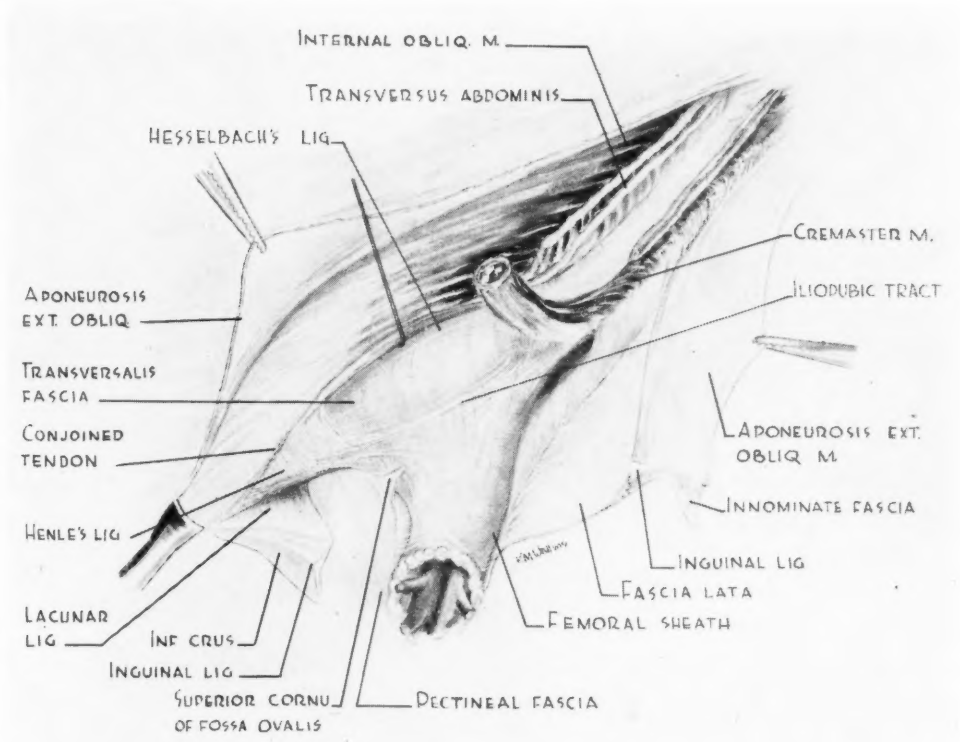

Fig. 3.—The transversalis fascia in the inguinal region. The relationship of the iliopubic tract, transversalis fascia, and the femoral sheath is shown. The inguinal ligament and fascia lata have been cut and reflected. The transected muscular portions of the internal oblique and transversus abdominis muscles are reflected to show the lateral portion of the transversalis fascia as it is continuous with the femoral sheath below. The separation of the conjoined tendon into its component parts (the transversus abdominis and internal oblique muscles) is shown. Henle's ligament is an inferior lateral expansion of the enveloping fascia and tendon of the rectus muscle, blending and continuous with the iliopubic tract and femoral sheath.

Surgically, Lotheissen in 1898 employed Cooper's ligament and the pectineal fascia in the cure of femoral hernia. Not until 1922, when Wise[15] furnished the germ for the idea, was the regular Bassini stitch carried very deep on through the inguinal ligament, so as to include a portion of the pectineal fascia. On the basis of this idea the following men approximated the lower end of the inguinal falx to the iliopectineal fascia and Cooper's ligament to obliterate the critical angle above the pubic bone: Andrews[1] (1924), Babcock[3] (1927), Dickson[5] (1936), and during the last decade, McVay and Anson[11] (1942), Neuhof[12] (1941), Harkins and associates[7] (1942), and most recently, Clark and Hashimoto[4] (1946).

In the more common types of inguinal herniorrhaphy such as advocated by Bassini, the medial portion of the transversalis fascia, commonly known as Hesselbach's ligament, and variously the transversus abdominis aponeurosis and the internal oblique muscle are sutured to the inguinal ligament, making a poor substitute for their normal insertion. The inguinal ligament is not the insertion of the transversalis fascia, transversus abdominis muscle, and the

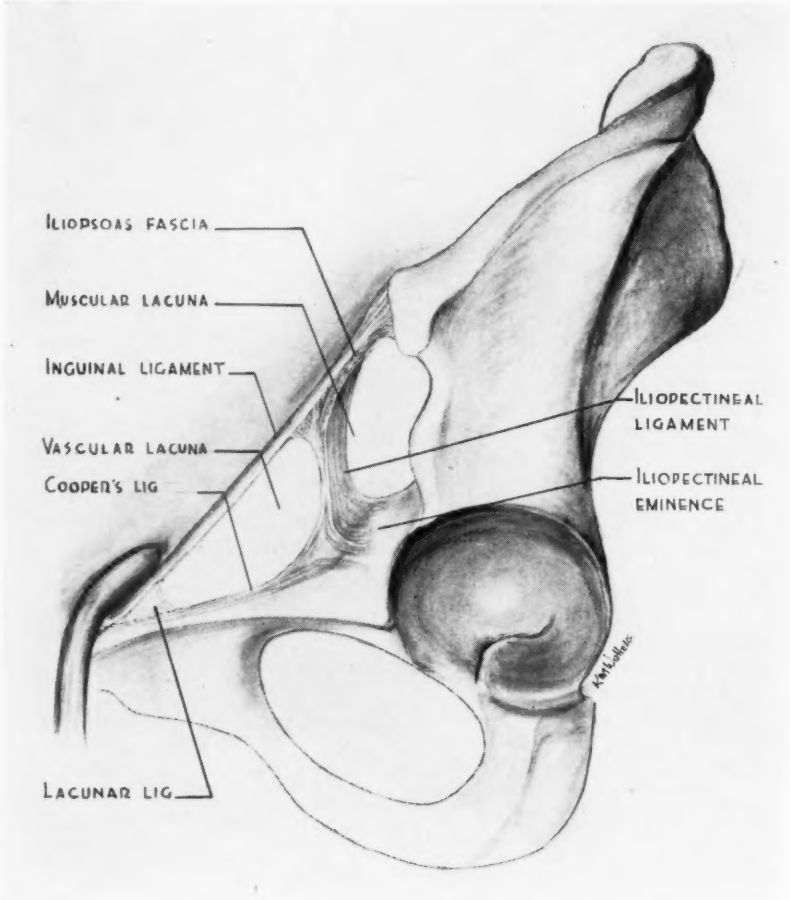

Fig. 4.—This diagram shows the location of Cooper's ligament, attached to the pubic bone inferiorly and to the iliopectineal eminence posteriorly, and its relationship to the inguinal and lacunar ligaments. The weak support of the inguinal ligament is apparent.

internal oblique muscle. Its relationship to these structures is merely one of proximity. Anatomically the inguinal ligament is weak in the low inguinal region. The only support it receives is from its insertion to the pubic bone medially, the anterior superior iliac spine laterally, and its attachment below to the fascia lata (Fig. 4). Surgically the inguinal ligament does not provide a suitable substitute for the insertion of these structures (the transversalis fascia, transversus abdominis, and internal oblique muscles), because it is composed of fibrous tissue. In the repair of large indirect, recurrent, and direct

hernias, it is recommended that the component parts of the transversalis fascia (Hesselbach's and Henle's ligaments and the iliopubic tract) be united with and anchored to Cooper's ligament, which is formed by the blending of these structures and the pectineal fascia onto the pubic bone. Cooper's ligament is the normal insertion, and, as McVay and Anson[11] (1942) have aptly put it, is "readily accessible, intrinsically strong, and directly fixed to bone."

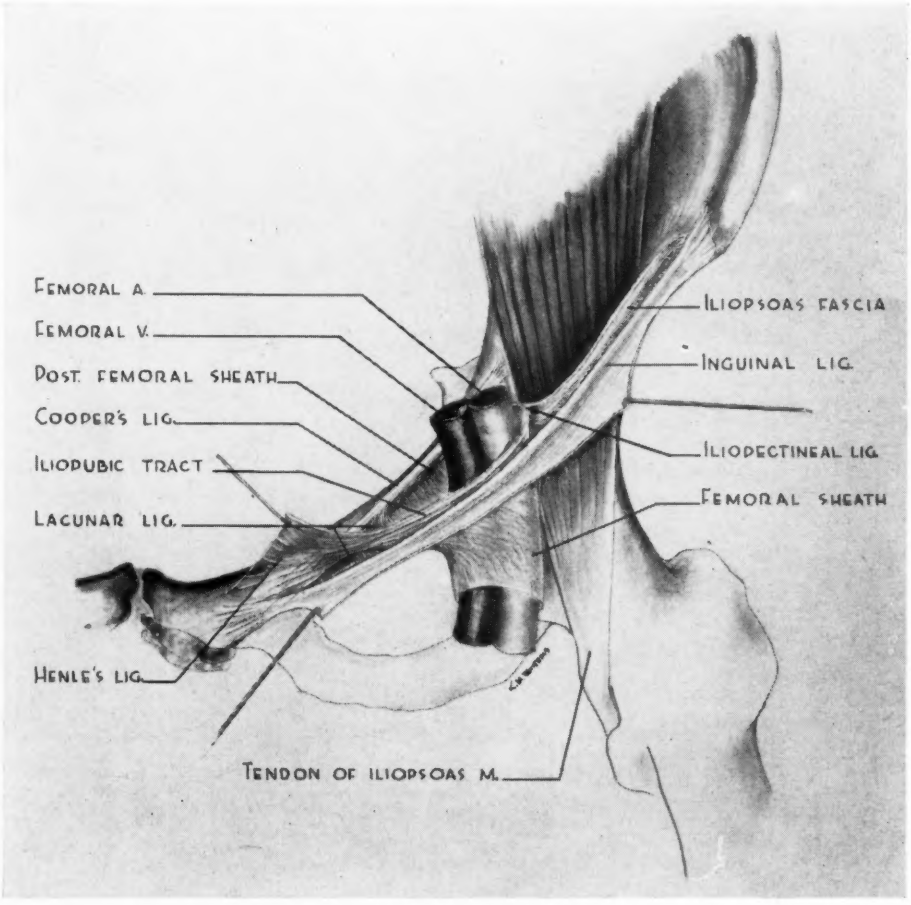

Fig. 5.—Deep bony and ligamentous structures of the inguinal region. This diagram shows the interrelations of Cooper's ligament (the superior pubic ligament), the lacunar ligament, Henle's ligament, the iliopubic tract, the iliopectineal ligament, and the inguinal ligament. The femoral vessels are displaced slightly laterally in order to clarify the structures about the femoral canal.

ANATOMY*

The transversalis fascia is a connective tissue or fascial membrane located between the peritoneum and the transversus abdominis muscle. It furnishes the chief fascial support for the anterior abdominal wall. In the inguinal

*The author wishes to express his appreciation to Dr. Robert Powers McBurney and Dr. William Robert Richardson for their assistance in cadaver and autopsy research; to Miss Katherine Morrow Watters for the original illustrations; and to Dr. J. O. Foley, head of the Department of Anatomy of the Medical College of Alabama, Birmingham, for valuable suggestions and advice concerning the anatomic features of this study.

region the transversalis fascia becomes thick and strong, reinforcing unprotected areas. Here the fibers assume a fan shape as they receive support at their different points of insertion. The handle portion occupies the upward position and the lower or open portion fastens onto the pubic bone and the different ligamentous tissues of this area. Along its medial portion it becomes blended with fascia of the rectus muscle and laterally to the dense iliopsoas fascia beneath the inguinal ligament. From the lower inguinal region it projects into the thigh with the femoral vessels as the femoral sheath. In the inguinal region

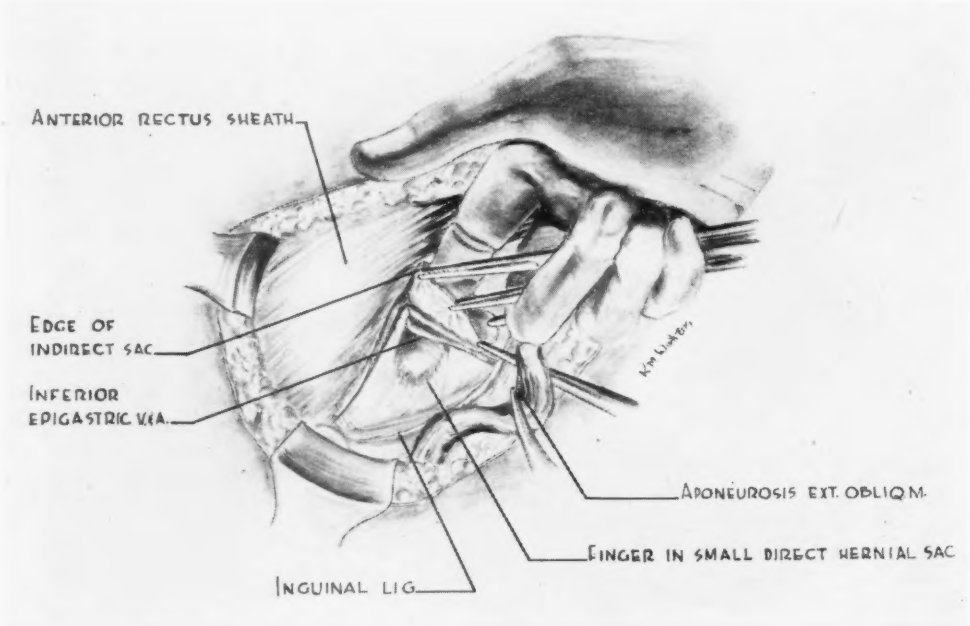

FIG. 6.—Dissection of the hernial sac for the diagnosis of direct and femoral hernia (after Fallis). The indirect hernial sac is opened and the index finger inserted into the peritoneal cavity. With this finger any small direct or femoral hernia can be demonstrated where it has not been recognized by routine examination. With clamps applied to the medial peritoneal leaf, a small direct or femoral hernia can be transposed by traction to become part of the indirect sac for purposes of repair (Hoguet's method). In large direct hernia, Hoguet's method is not applicable (see text).

the transversalis fascia is potentially weakened in two areas: the upper or midportion of the fascia is pierced by the internal abdominal ring, and the lower portion, above the pubic bone, in the unprotected area commonly called the Hesselbach space, does not receive support from the internal oblique and the transversus abdominis muscles. As a result of its modifications in the inguinal region, the following component parts of the transversalis fascia are derived: the ligament of Hesselbach (ligament interfoveolare), Henle's ligament, and the iliopubic tract.

Ligament of Hesselbach or the Ligament Interfoveolare.—The ligament of Hesselbach is located just lateral to the inferior epigastric vessels and along the medial margin of the internal inguinal ring in the inguinal space (Figs. 3 and 7). Its fibers are chiefly vertical, and it is triangular in shape, due to its attachment above and fixation below. Externally it receives fibers from the trans-

versus abdominis aponeurosis. It is inserted below at the pecten of the pubes, where its fibers fuse into Cooper's ligament, and in its upper insertions its fibers pass laterally to the internal abdominal ring, medially across to the sheath of the rectus muscle, and finally extend to the secondary arcuate line which lies below the fold of Douglas.

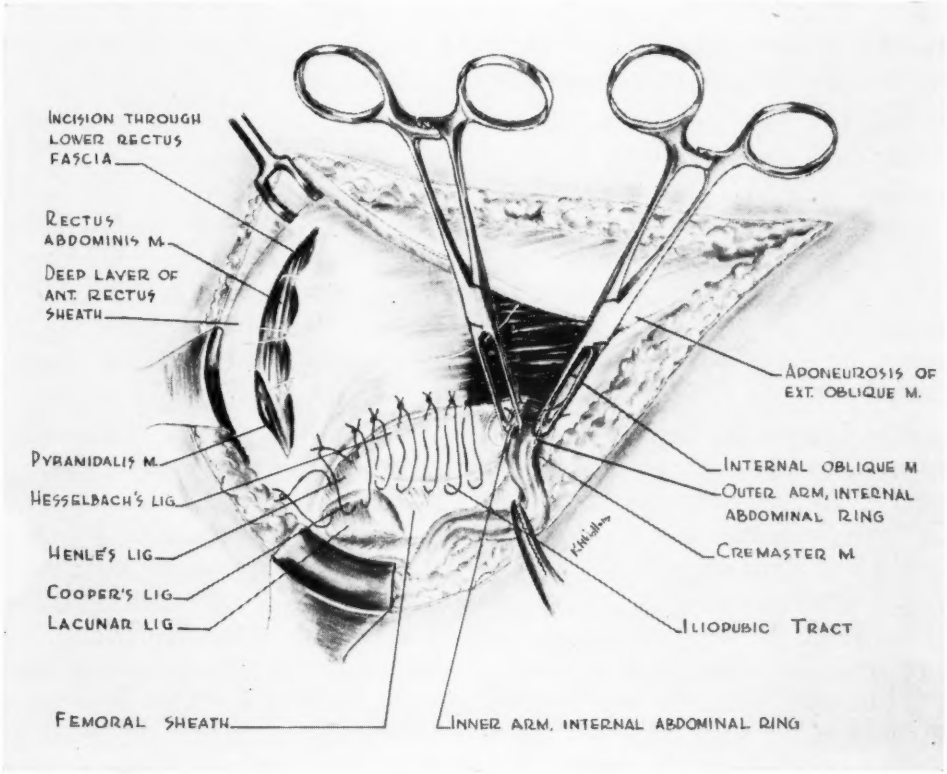

Fig. 7.—First row of sutures. Three sutures are placed, 1 to 2 cm. apart, through Hesselbach's ligament and Henle's ligament above and Cooper's ligament and the lacunar ligament below, including the medial part of the iliopubic tract. The third suture is the last one incorporating Cooper's ligament. The remaining sutures approximate Hesselbach's ligament to the iliopubic tract up to the level of the internal abdominal ring. In cases in which Hesselbach's ligament above is thin, the aponeurosis of the transversus abdominis muscle is included in the suture bite. In this drawing a vertical relaxing incision has been made in the part of the anterior rectus fascia posterior to the external oblique aponeurosis. The inguinal ligament is not shown. The two arms of the internal abdominal ring have been identified and brought into view with Allis clamps preparatory to their approximation with interrupted sutures above the spermatic cord.

Ligament of Henle.—Henle's ligament lies in the floor of the inguinal space (Figs. 2, 3, and 5). Its fibers run chiefly in the transverse direction. It is somewhat triangular in shape and concave above. Its base is inserted onto the pubic bone. Its outer fibers blend with the iliopectineal fascia, the base of Cooper's ligament, and the base of Hesselbach's ligament. Along its medial surface it receives fibers from the lowermost portion of the rectus fascia.

The Iliopubic Tract or the Ligament of Thompson.—The iliopubic tract forms the outer leaf of the transversalis fascia (Figs. 3, 5, and 7). It lies beneath the inguinal ligament. Medially it swings posteriorly and attaches to

the pecten of the pubes, in company with the outer leaf of Henle's ligament, the base of Cooper's ligament, and the iliopectineal fascia. Laterally it is attached to the anterior superior iliac spine, where its fibers spread like a fan in a divergent manner, some going to the spine itself and others onto the internal lip of the iliac crest. It receives additional support from the femoral sheath and the iliopsoas fascia, which is partially evident as it crosses the femoral vessels. Roughly its fibers are both vertical and transverse. Many of the transverse fibers cross the inguinal space, blending with Hesselbach's ligament, and with the lateral border of the adminiculum lineae albae.

Cooper's Ligament or the Superior Pubic Ligament.—Cooper's ligament extends from the tubercle of the pubis along its superior border to the iliopectineal eminence (Figs. 4, 5, and 7). It is formed by the fusion of the pubic attachments of the three component portions of the transversalis fascia (Hesselbach's ligament, Henle's ligament, and the iliopubic tract), and the pectineal fascia. It appears as a thick, fibrous cord intimately adherent to the bone and runs posteriorly at an angle of 30 to 35 degrees to the inguinal ligament. Because of its strength and position its utilization in the repair of inguinal hernia is very important.

The Internal Abdominal Ring.—The internal abdominal ring, as described by Lytle,[10] stands out as the pivot around which the transversalis fascia is arranged, located midway between the anterior superior spine of the ilium and the pubis, approximately 2 cm. above the inguinal ligament (Fig. 7). It is made up of sling fibers derived from the transversalis fascia and attached to the undersurface of the transversus abdominis muscle. The ring is not round, but is U-shaped. Its open ends point upward and the angle of the ring points downward. The fibers of the angle of the ring are much shorter than the fibers of either arm. The fibers of the medial arm of the ring are much longer than the fibers of the outer arm, and often the fibers of the medial arm are found attached as far up as the arcuate line or the fold of Douglas. The outer arm fibers, being shorter and smaller, find their attachment to the transversus abdominis muscle in proximity to the anterior superior spine of the ilium. This arrangement of the ring fibers gives a freer movement of the internal arm when the abdominal muscles are brought into action by the different exercises of these muscles and produces a somewhat sphincter-like action of the abdominal ring. (For further details of the anatomy and the physiology of the internal abdominal ring, consult Lytle.[10])

The Inguinal Space.—The inguinal space, or outer suprapubic space, is the space that is not covered by the transversus abdominis and internal oblique muscles (Figs. 2 and 3). It is in this space that the transversalis fibers are the weakest and usually its fibers here run transversely. It represents the dome portion of the transversalis fascia, with the weakest support, commonly called Hesselbach's triangle, the base of which is formed by the medial portion of the inguinal ligament, the medial side by the lateral border of the rectus abdominis muscle, and its lateral border by the inferior epigastric vessels. It is through this space that direct inguinal hernias occur. This space is divided into the

middle inguinal fossa and the internal inguinal or supravesicular fossa, and it is through the middle inguinal fossa that the majority of direct inguinal hernias occur. Rarely does the hernia originate in the internal inguinal fossa, which lies medial to the obliterated umbilical artery; here the transversalis fascia gets support from the rectus muscle and the conjoined tendon. In dealing with a large direct inguinal hernia it is very important after the hernial sac has been dealt with to close the opening in the transversalis fascia through which the hernia made its exit, which closure is independent of the support obtained by approximating the different component portions of the transversalis fascia and utilizing Cooper's ligament.

SURGERY

Spinal anesthesia is used unless contraindicated, when local anesthesia is employed. For the local anesthesia a 1 per cent solution of Novocain and 3 drops of adrenalin chloride to the ounce are employed. The objection to local anesthesia in repair of inguinal hernia is the time consumed in its injection. It usually requires thirty to forty minutes longer. In the inguinal region a complete block anesthesia from local Novocain injection, however, can be obtained with complete relaxation of the tissues with the minimum amount of solution.

The Bassini incision is employed. The incision should not extend onto the scrotum or the low pubic area, as the skin in these areas harbors bacteria and if these tissues are incised it may lead to infection of the wound. In the only two cases of infection in this series, both occurred where incisions had been extended onto these areas. With a blunt retractor placed in the lower angle of the wound, sufficient exposure of the deeper structures can be obtained without incising the low pubic or scrotal area. In cases of hernia the superficial vessels of the lower angle of the wound often are larger and more numerous than in the upper angle, possibly due to the irritation from the hernial bulge or pressure from a truss. To maintain hemostasis the larger vessels should be carefully dissected out and doubly clamped before they are divided. The smaller vessels and the capillaries should be clamped and ligated. This prevents hematoma and minimizes serum accumulation, thus lessening the danger of infection and interference with tissue repair.

The suture material is either cotton or silk. A heavy grade is used for the deep sutures, medium for the external aponeurosis and Scarpa's fascia, and fine for hemostasis. The chief value of nonabsorbable material over surgical suture (catgut) is that it maintains apposition of the tissues until union occurs, and lessens tissue reaction for serum accumulation, further reducing the risk of infection.

The skin incision is carried through Scarpa's fascia to the aponeurosis of the external oblique muscle (Fig. 1), where sponge dissection should be employed to clear away the areolar and adipose tissues from the surface of the aponeurosis throughout the incision. The aponeurosis is opened by a small incision in the upper angle of the wound. With elevation of the margins of the aponeurosis, the incision is carried through the fibers down to and including the substaneous

ring. The ring is opened along its upper medial surface, bringing the ilioinguinal and iliohypogastric nerves into view (Fig. 2). The ilioinguinal nerve runs parallel to and medial with the spermatic cord. Opposite the subcutaneous ring the ilioinguinal nerve divides into several branches, some of which often pierce the ring fibers of the external oblique aponeurosis. Good exposure prevents injury to these nerve fibers when the ring is opened. The iliohypogastric nerve makes its superficial exit several centimeters above the bed of the ilioinguinal. It runs in a transverse direction and pierces the rectus fascia along the outer margin. Injury to either nerve may result in weakening the muscles, which may encourage a recurrence of the hernia. Also, injury to the ilioinguinal nerve and its fibers at the subcutaneous ring will produce a numbness of the scrotum and medial skin surface of the thigh.

The ilioinguinal nerve is further protected by being dissected from its bed and withdrawn in an outward lateral manner. The spermatic cord is separated from its bed of adhesions and is withdrawn in a like manner. In many cases the hernial sac is of such size that it fills the entire canal. It is difficult in such cases to determine whether the hernia began as an oblique or a direct, since there is a giving way of the entire transversalis fascial support. The deep epigastric vessels which normally run inferior and medially to the internal abdominal ring no longer serve as a guide for diagnosis between an oblique and a direct hernia. When the hernial bulge attains large size these vessels are sometimes displaced to a position above and opposite the internal abdominal ring. The diagnosis of small direct or femoral hernia suggested by Fallis[6] constitutes a real advance in the recognition of these hernias when they have not been observed by routine examination. Fallis suggested, in cases of small direct hernia, opening the peritoneum at the internal abdominal ring above and medial to the spermatic cord. The surgeon's finger enters the peritoneal cavity and the entire inguinal space, including the femoral region, is palpated for weak points and peritoneal pouching (Fig. 6).

In the management of small direct hernias, or cases where the dome portion of the transversalis fascia is herniated, when they accompany oblique hernia, their treatment should be carried out in the fashion recommended by Hoguet.[9] However, for the large direct hernia with definite sac formation, the Hoguet method is not applicable. This type of hernia is treated in the same way as an oblique hernia. This is accomplished by freeing the hernial sac of adhesions down to the abutment of the peritoneum. In both large direct and oblique hernias, the hernial opening in the peritoneum is closed by purse-string suture placed on the inside of the sac at its base. An alternate for the purse-string suture is the transfixed stitch placed at the base of the open sac. In the large direct hernia, the hernial ring in the transversalis fascia must be closed independently of the reconstruction of the fascia.

The practice of anchoring the stump of the sac to the undersurface of the overlying muscles in the oblique hernia has been advocated by some as the best method of preventing recurrence of hernia. It has been observed that this treatment may lead to the pocketing of the peritoneum at the point of anchorage

and invite recurrence. Passing the suture through and over the muscles may also interfere with the action of the muscles and weaken them by strangulation.

The complete removal of the hernial sac and closure of the internal abdominal ring are essential for a successful operation for oblique hernia. In the closure of the ring both arms and the angle of the ring should be identified. This can be done by elevating the spermatic cord and freeing adhesions beneath the cord by sponge dissection down to the angle of the ring. The angle of the ring will appear as dense fibers firmly adherent to the bed of the transversalis fascia. From this point each arm of the ring will be seen to run in line with its normal position. To facilitate the exposure of each arm, an Allis clamp may be placed on its fibers, and with elevation of the clamp the respective arms will come into view (Fig. 7). After such exposure the arms of the ring are approximated by two to three interrupted sutures above the cord. In placing the sutures one should avoid incorporating the spermatic cord tissues or closing the ring so tightly as to constrict the cord.

After the hernial sac and the hernial ring have been properly treated, it is necessary to clear the inguinal canal of areolar and adipose tissue, accessory vessels, and straggling muscle fibers. Most frequently the accessory vessels are found beneath the cord as it swings over the pubic bone. Often the vessels are of large size and they should be doubly clamped before dividing. In the recurrent hernia varying fibrous bands of tissue will be found adherent to the inguinal ligament from the previous approximation of the supportive structures to the ligament. The inguinal ligament is to be freed of the fibrous tissue. This affords good visualization of the operative field, including the component parts of the transversalis fascia and Cooper's ligament. Cooper's ligament may be identified as a fibrous cord by palpation of the superior margin of the pubic bone. It is covered by the thin pectineal fascia, and it can be readily discerned by placing an Allis clamp onto the pectineal fascia so as to include the ligament.

For statistical purposes the size of the hernial cleft may be obtained by a linear and transverse measurement of the cleft, using a graduated uterine probe.

Upon examination, the hernial cleft may be found to be of such size that, with retraction of the Hesselbach ligament and the aponeurosis of the transversus abdominis muscle, approximating these structures to the iliopubic tract and Cooper's ligament may lead to tension along the suture line. In such cases, Rienhoff and others have suggested incising the rectus fascia at its pubic attachment, extending the incision in an upward manner for several centimeters (Fig. 7). This will provide relaxation of these structures for normal tissue closure. It has been found that such a procedure will not weaken the rectus muscle and the pyramidalis muscle support. However, in only one case in this series was a relaxing incision in the rectus fascia found necessary, due to atrophy of Hesselbach's ligament and the transversus abdominis aponeurosis following a previous operation for hernia.

Closure of the hernial cleft should be begun at the lower angle of the wound over the pubic bone. Harkins and Swenson[8] preferred placing the first suture several centimeters above the pubic bone in proximity to the femoral vein to lessen the chance of injury to the vein in placing a suture. I recommend

commencing the suture line over the pubic bone and passing the first suture through the lowermost portion of Hesselbach's ligament, Henle's ligament, and the iliopubic tract, including Cooper's ligament and fibers of the lacunar (Gimbernat's) ligament (Fig. 7). It was found that this procedure accomplishes two things: First, the iliopubic tract at its pubic insertion is often small and obscure, lying beneath the inguinal ligament. The elevation of the iliopubic tract by the suture will better identify the tract. Second, there is a better coaptation of the ligamentous structures. Occasionally, the iliopubic tract at its pubic insertion may be so obscure that it is difficult to recognize its fibers for placing the first suture. In such cases, before the suture line is commenced, the iliopubic tract can be brought into clear view by passing the blunt end of the scalpel handle beneath the inguinal ligament opposite the pubic bone to separate the loose adhesions between the inguinal ligament and the iliopubic tract. In this way the aponeurotic fibers of the iliopubic tract will be clearly differentiated from the white fibers of the inguinal ligament. Utilizing the iliopubic tract gives essential lateral support to the transversalis fascia.

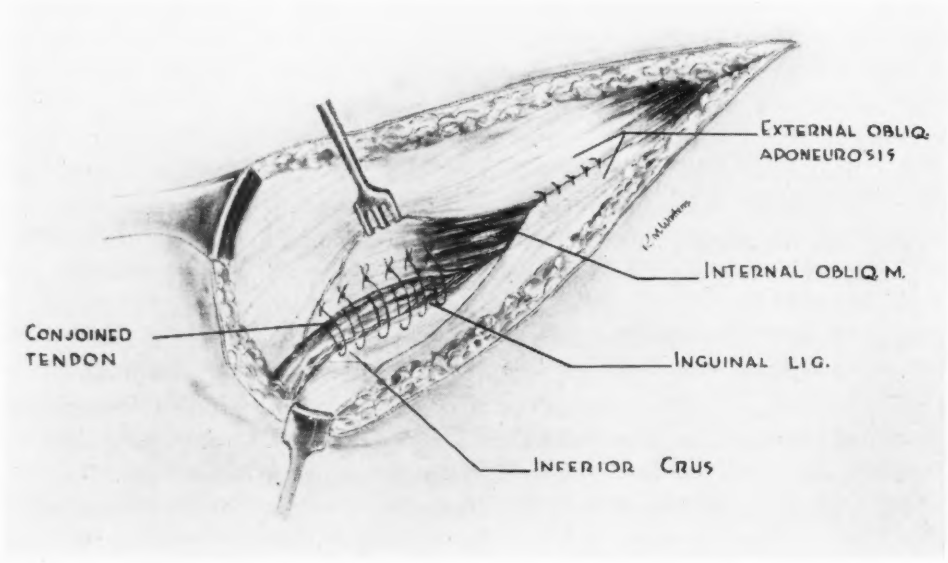

Fig. 8.—Second row of sutures. The spermatic cord is replaced in its normal position, resting on the first row of sutures. The fibers of the conjoined tendon are loosely approximated to the inguinal ligament above the cord. The extent of the approximation of the conjoined tendon to the inguinal ligament depends on the condition of the muscle fibers in the low inguinal region (see text). Shown also is the beginning of the closure of the external oblique aponeurosis, with interrupted sutures.

The sutures are placed 1 to 2 cm. apart, and upon placement of the third suture, which is the last one incorporating Cooper's ligament, attention is given to the femoral vessels (Fig. 7). To avoid injury to these vessels it is necessary for the surgeon to place his index finger into the wound and retract the vessels in a lateral manner before the suture is inserted. The suture line for approximating Hesselbach's ligament and the transversus abdominis muscle to the iliopubic tract is continued in an upward manner to the level of the internal abdominal ring (Fig. 7).

Testut and Jacob,[14] in their anatomic description of the iliopubic tract, and McVay and Anson,[11] discussing the clinical use of the iliopubic tract, have spoken of the diminution in the size of the tract as it crosses the femoral vessels (Fig. 5). It has been found, however, that the iliopubic tract is not altered at this point to such an extent as to interfere with approximation of the tract to Hesselbach's ligament and the transversus abdominis aponeurosis.

The operation is completed as follows: The spermatic cord and the ilioinguinal nerve are replaced to the inguinal bed and permitted to rest on the suture line following the closure of the transversalis fascia (Fig. 8). The open or dead space above the pubic bone, bounded laterally by the spermatic cord and the inguinal ligament, medially by the rectus fascia below, and above and medially by the internal oblique and transversus abdominis muscles, is dealt with in the following manner: First, if the case is one in which there is a congenital absence of the internal oblique and transversus abdominis muscles in the low inguinal space, or atrophy of these muscles from disuse, nutritional deficiency, or following necrosis secondary to previous surgery, no attempt is made to close the space except loosely to approximate the muscle fibers of the internal oblique and transverse abdominis muscles to the inguinal ligament, above the cord, opposite the internal abdominal ring. Second, if the internal oblique and transversus abdominis muscle fibers are healthy and well developed, they are loosely approximated to the inguinal ligament, above the cord, opposite the internal abdominal ring as far downward as these muscle fibers extend. In closure of the fibers of the external oblique aponeurosis and the reconstruction of the subcutaneous ring, care should be taken to avoid constriction of the spermatic cord by sutures (Fig. 8). Closure of Scarpa's fascia and the skin completes the operation.

SUMMARY

1. The recurrences following the standard (Bassini) operation for large oblique and direct hernias prompted the employment, instead, of an operation utilizing the component parts of the transversalis fascia and allied ligamentous structures in such cases. In 116 cases of large oblique, recurrent, and direct hernias in which this procedure was employed, questionnaires sent to the patients showed that there has not been a single recurrence of hernia, with all questionnaires answered except three, which were returned as unclaimed.

2. To further the knowledge of the deep supportive structures of the inguinal region, an anatomic review has been made of these structures. This has consisted of a review of the literature in this field, cadaver research, autopsies, and clinical observations at operations.

3. The pathology of all inguinal hernia is not the same. To obtain the maximum results in the operation for inguinal hernia, it is necessary to consider the pathologic changes that accompany the different types of hernias in the inguinal supportive tissues. Since the transversalis fascial support is not involved in the small oblique hernia, usually seen in children, the treatment consists simply of the removal and closure of the patent processus vaginalis (sac) down to the fold of the peritoneum and the closure of the internal abdominal ring, as is employed in the Bassini operation.

4. The chief pathologic features in large oblique and direct hernias are the giving way of the transversalis fascial support and varying degrees of dilatation of the internal abdominal ring. Operations for these hernias should be directed toward restoring these structures to a normal state.

5. Special emphasis is placed on the importance of including the iliopubic tract in the suture line for closure of the transversalis fascia to maintain the lateral support of the fascia. In the oblique hernia, the dilated internal abdominal ring should be closed by identifying its arms and approximating them with interrupted sutures above the spermatic cord.

6. In the operative management, care should be taken to maintain hemostasis, thus preventing hematoma and serum accumulation which might invite infection and weaken the tissue support, and to avoid unnecessary trauma of the tissues, including the nerve supply, which might result in weakened muscles. The operative field should be cleared of areolar and adipose tissues and straggling muscle fibers for good visualization and prevention of their incorporation in the suture line, which would weaken the ligamentous support.

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A DEFINITIVE AMBULATORY TREATMENT FOR INFECTED PILONIDAL CYSTS

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(From the Cleveland Clinic)

OPERATIONS for the cure of pilonidal cysts fall into two broad classifications: (1) excision of the cyst with or without closure; (2) marsupialization of the cyst with suture of the skin to the epithelium at the base of the cyst.

Excision of the cyst with primary closure is not applicable to acutely infected cysts because contamination and sacrifice of tissue results in a large infected dead space. Excision of the cyst and packing it open results in slow healing, and when the buttocks are fat it may be as long as a year before epithelization is complete. Moreover, the ensuing scar is broad and is often painful and breaks down if traumatized. Marsupialization of the cyst with suture of the skin to the base sacrifices no tissue and results in faster healing. Even with this technique, however, the resultant wound takes weeks or even months to heal, requires many dressings, and causes prolonged disability.

The original epithelial rest is small and causes difficulty only because the desquamated epithelium has no way of being discharged externally. To make an incision several inches long in order to marsupialize and provide drainage for a tiny epithelial rest seems unnecessarily radical. Before the cyst is distended by acute infection the epithelial rest is measurable in millimeters rather than in inches. If marsupialization could be effected by a conservative office procedure, and if following this the patient could be ambulatory and remain at work, a great economic saving would be effected.

The first patient that I treated by this method was a Wave in the U. S. Navy Hospital Corps who had had three operations for a pilonidal cyst and developed an acutely infected recurrence. She did not wish to enter the hospital and would not consent to a further attempt at excision of the cyst. I therefore incised and drained the abscess and left a short section of a mushroom catheter in the cavity to maintain drainage.

The patient continued to perform her duties on the ward and tolerated the mushroom catheter without discomfort. Since the abcess was large and the buttocks were fat, I hesitated to remove the catheter too soon. After a month it became apparent that epithelium was growing down the sides of the opening, and by the end of three months epithelization of the tract appeared to be complete. In the meantime the cavity was no larger than the mushroom of the catheter. There was no significant drainage. The catheter was removed, and within a few days the cavity had filled in and epithelized to such an extent that the only visible remnant was a deep, completely epithelized dimple about 4 mm. in diameter, the base of which appeared to be on the periosteum of the sacrum. Follow-up was carried out on the patient for one year, during which time there was no recurrence.

Since 1944 I have treated twelve patients with acutely infected pilonidal cysts by this technique with uniform success. There have been no recurrences and no complications. The retention catheter has caused no discomfort or disability. I have also tried to treat pilonidal sinuses by this technique but with

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only indifferent results. It is difficult to get the tube in place and to maintain it in the proper position, and I doubt that the results will prove satisfactory. It is the acutely infected cyst that is ideally adapted to this technique.

TECHNIQUE

The technique is illustrated in Figs. 1 to 4. The steps are as follows:

1. The skin is frozen with ethyl chloride.

2. An incision about 1½ cm. in length is made in the midline directly over what is estimated to be the exact center of the cyst.

3. The contents of the abscess are evacuated, and a mushroom catheter is inserted.

4. A safety pin is put through the catheter to prevent it from falling into the cavity, and the tube is cut off distal to the pin.

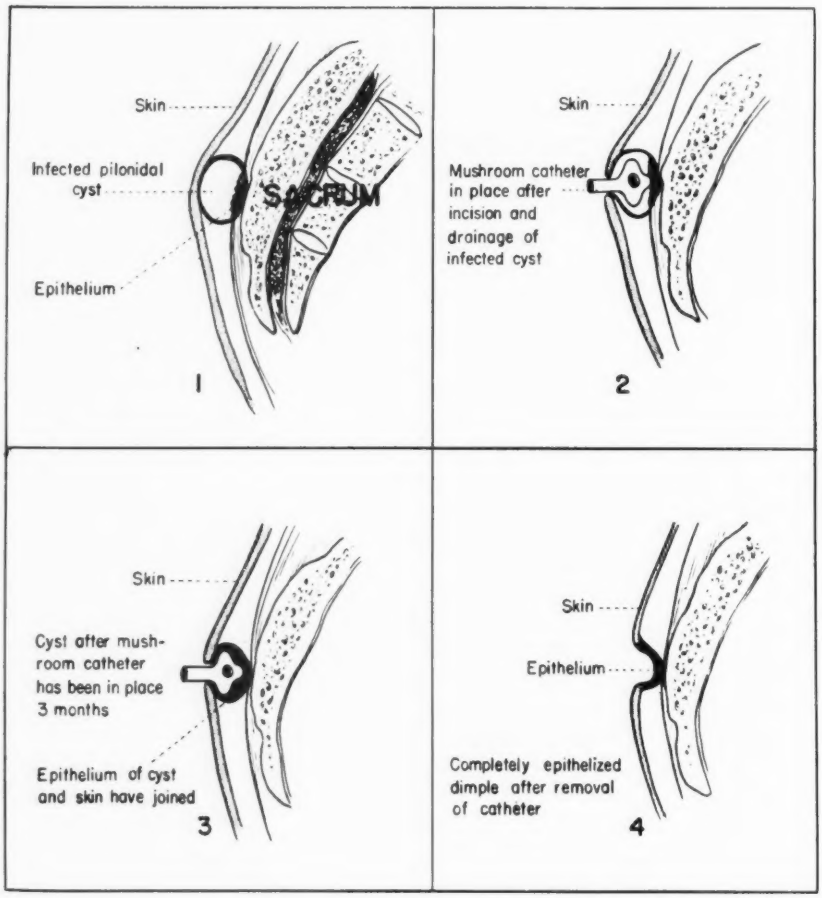

Fig. 1.—Pilonidal cyst; note epithelium at bottom of cyst.

Fig. 2.—Infected cyst after incision and drainage with mushroom catheter in place.

Fig. 3.—Cyst after mushroom catheter has been in place three months; note epithelium of skin has met that of cyst.

Fig. 4.—Completely epithelized dimple after removal of catheter.

5. The patient is instructed to take sitz baths twice daily until the discharge diminishes.

6. At the end of one week the catheter is shortened and the safety pin is removed. The cavity by this time has diminished in size to such an extent that the catheter can be cut flush with the skin without danger of its falling back into the cavity.

7. The patient requires no further dressings or treatment for one month, at which time the catheter is again shortened by cutting it off flush with the skin.

8. At the end of three months the epithelium has grown down along the catheter and presumably has joined the epithelium growing in the bottom of the cyst, thus marsupializing the cyst. The catheter is therefore removed.

9. Inspection at the end of one week shows nothing more than a deep dimple with its epithelized base resting on the periosteum of the sacrum. The patient is discharged.

From the economic standpoint this treatment has the following advantages: (1) no hospitalization, (2) no operation other than a simple office procedure, (3) only four postoperative office calls, (4) no dressings after the first week, and (5) no loss of work after the first day.

From the standpoint of cure of the disease this treatment has the following advantages: (1) The epithelial rest apparently is completely and permanently marsupialized; (2) there are no painful dressings; (3) there is no deformity or scar other than a small dimple.

SUMMARY

A simple office procedure for the definitive treatment of acutely infected pilonidal cysts is described.

ISCHIAL DECUBITUS ULCER

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AMONG the recent references which deal with the management of decubitus ulcer[1, 3-6, 8-14, 16-18, 22, 31, 32] there are only four[5, 9, 18, 32] which mention the treatment of ischial bedsores. Since the incidence of ischial decubitus ulcers increases in proportion with the number of patients reaching the ambulatory stage it appeared worth while to report our experience in forty-one patients.

INCIDENCE, DISTRIBUTION, SECONDARY BONE CHANGE, SIZE

Forty-seven ulcers were present in forty-one patients with spinal cord injuries. Among them there were only three cases not due to war wounds or war accidents. One cord injury developed after spinal anesthesia, another consequent to meningitis and epidural abscess, and a third was caused by a cord tumor.

Table I illustrates the level, date of injury, number of sores, types of intervention, and results in the respective cases.

The shortest interval before a sore developed was three months and the longest was thirteen years. The duration of the sore prior to operation ranged from one month to three years.

There were two cervical, sixteen upper dorsal (first to sixth), seventeen lower dorsal (seventh to twelfth) and six lumbar lesions. No relationship could be established between the level of the lesion and the secondary bone changes. According to x-ray and biopsy findings bone involvement was present (Figs. 1 and 2) in thirty-five cases. The size of the ulcer varied from 2 by 4 cm. to 8 by 10 cm.

ETIOLOGY

The etiology of decubitus ulcers has recently been discussed at length.[9, 17, 18, 27] It has been pointed out that general factors such as hypoproteinemia,[3, 4, 25, 27] exhaustion,[27] and flare-up of infections[27] play a major role. However, as to the local development there are still two schools of thought: simple local trauma by pressure versus trophic changes. The first version has been upheld by Freeman[17] and Gibbon and Freeman.[18] Freeman[17] saw an equal timed response to histamine flare, wheel, and tache in the skin above and below the cord lesion in all patients. T. B. Massell (personal communication of unpublished data) observed good circulation in granulations by the intravenous fluorescin method in six cases of cord injury with decubitus ulcers at DeWitt General Hospital, Auburn, Calif., in 1945. These findings are in contrast to the theory of locally disturbed sympathetic reflexes as an additional cause for the development of decubitus ulcer.[27] It is evident that the neurogenic factor plays an important role. It is responsible for loss of sensation, motor paralysis, and consequent disuse atrophy with decreased circulation of the subcutaneous

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structures.[17] But the circulation of the skin itself does not seem to be impaired. Wherever continuous weight-bearing exerts pressure on a poorly padded skin area breakdown of the skin will ensue. This applies particularly to the ischial region. The majority of our patients displayed an atrophy of the buttocks. The precipitation of the sores was caused by prolonged sitting, by sitting in wet wrinkled clothes, or another local trauma. In contradistinction to the development of other ulcers after a "sore-free interval," the ischial ulcer does not

Fig. 1.—B. H. S., aged 24 years, level of the fourth dorsal vertebra, date of injury April 2, 1945. Bilateral ischial decubitus ulcer with osseous involvement, calcification, and histologically proved bony metaplasia of bursa on left prior to operative repair.

reflect imbalance of the patients' health. Such ulcers develop by mere trauma in the absence of protein disturbances, anemia, or infection. It is furthermore obvious that ischial ulcers are preceded by a "sore-free interval" because there is no weight-bearing, favoring their development.

ANATOMY, PATHOLOGY, HISTOLOGY, GROSS APPEARANCE

The anatomy[7, 14, 26] of the ischial region reveals the presence of bursae. Some authors describe the location of bursae as higher than the real point of

TABLE I

	PRIMARY METHOD														SECONDARY METHOD							
NUMBER	NUMBER OF ULCERS	INITIALS OF PATIENTS	AGE OF PATIENTS	DATE OF INJURY	LEVEL OF INJURY	COMPLETE OR INC.	SIDE (RIGHT OR LEFT)	SECONDARY BONE CHANGES	BURSA	NUMBER OF CLOSURES	RESULT OF LAST CLOSURE	TIME SINCE HEALING	RECURRENCES	NUMBER OF ULCERS	SECONDARY BONE CHANGES	BURSA	NUMBER OF CLOSURES	RESULT OF LAST CLOSURE	TIME SINCE HEALING	RECURRENCES	NUMBER OF ULCERS
1	1	S. S.	22	1/45	D-12	Comp.	Right	Yes	Yes	1	Exc.	?	Yes	1	Yes	No	1	Exc.	5 mo.	No	1
	2						Left	Yes	Yes	1	Good	?	Yes	2	Yes	Yes	1	Exc.	6 mo.	No	2
2	3	L. S.	24	8/29/43	D-4	Comp.	Right	Yes	Yes	1	Exc.	2 yr.	No	3							
3	4	P. L.	21	10/3/44	D-4	Comp.	Left	Yes	Yes	1	Exc.	1 yr.	No	4							
4	5	S. M.	29	9/4/44	L-1	Inc.	Right	No	No	1	Exc.	1 yr.	No	5							
	6						Left	Yes	Yes	3	Poor			6	Yes	Yes	1	Exc.	4 mo.	No	3
5	7	H. M.	26	10/6/44	D-4	Comp.	Right	Yes	Yes	2	Poor			7							
6	8	J. P.	20	5/22/45	L-2	Comp.	Right	No	Yes	1	Exc.	1 yr.	No	8							
7	9	H. R.	20	4/1/45	D-6	Inc.	Right	No	No	1	Good	16 mo.	No	9							
8	10	D. D.	28	3/8/46	L-2	Inc.	Right	No	Yes	1	Exc.	6 mo.	Yes	10	No	No	1	Exc.	5 mo.	No	4
9	11	E. K.	43	6/7/31	D-11	Comp.	Left	No	Yes	1	Exc.	13 mo.	No	11							
10	12	E. S.	29	2/42	D-4	Comp.	Right	No	Yes	1	Exc.	1 yr.	No	12							
11	13	D. L.	26	4/3/45	L-1	Inc.	Left	No	No	2	Poor			13	Yes	No	2	Poor			5
12	14	C. M.	29	9/17/44	D-9	Comp.	Right	No	No	1	Exc.	1 yr.	No	14							
13	15	M. T.	29	6/1/44	D-11	Comp.	Right	No	No	1	Exc.	1 yr.	No	15							
14	16	G. L.	40	7/26/44	D-12	Comp.	Right	No	Yes	1	Exc.	13 mo.	No	16							
15	17	E. D.	33	6/4/45	D-4	Comp.	Left	No	Yes	2	Exc.	10 mo.	No	17							
16	18	C. H.	26	11/11/42	D-11	Inc.	Left	Yes	Yes	2	Exc.	8 mo.	Yes	18							
17	19	B. S.	24	4/2/45	D-4	Comp.	Right	Yes	Yes	1	Exc.	10 mo.	No	19							
	20						Right								Yes	Yes	1	Exc.	6 mo.	No	6

18	21	A. L.	21	3/13/44	D-8	Comp.	Right	Yes	Yes	1	Exc.	10 mo.	No	20						
	22						Left	Yes	Yes	1	Exc.	10 mo.	No	21						
19	23	R. C.	23	6/13/44	D-3	Comp.		Yes	Yes	3	Exc.	4 mo.	Yes	22	Yes	Yes	1	Exc.	1 mo.	No	7
20	24	J. H.	22	10/20/45	C-6	Inc.	Right	No	No	1	Exc.	1 mo.	No	23							
21	25	M. I.	55	2/1/41	D-12	Comp.	Right	Yes	Yes	1	Exc.	3 mo.	No	24							
22	26	H. B.	25	5/30/43	C-6	Inc.	Left								Yes	No	1	Exc.	4½ mo.	No	8
23	27	R. B.	23	4/22/45	D-11	Comp.	Left								Yes	Yes	1	Good	3 mo.		
24	28	L. D.	26	9/20/44	D-3	Comp.	Right								Yes	Yes	1	Good	6 mo.	No	10
25	29	A. E.	21	6/6/45	D-11	Comp.	Left								Yes	Yes	2	Exc.	1 mo.	No	11
26	30	L. L.	23	9/18/44	D-9	Comp.	Left								Yes	No	1	Exc.	5 mo.	No	12
27	31	R. L.	26	3/21/45	D-1	Comp.	Left								Yes	Yes	1	Exc.	2 mo.	No	13
28	32	J. L.	31	5/12/43	D-10	Comp.	Left								Yes	Yes	1	Exc.	2 mo.	No	14
29	33	J. M.	27	7/11/44	D-9	Comp.	Left								No	Yes	1	Exc.	6 mo.	No	15
30	34	R. M.	26	8/31/45	D-4	Comp.	Right								Yes	Yes	1	Exc.	3½ mo.	No	16
31	35	R. N.	23	11/9/45	Cauda equina	Inc.	Right								Yes	Yes	1	Exc.	?	Yes	17
32	36	N. O.	26	12/1/45	D-10	Comp.	Right								Yes	Yes	1	Poor			18
33	37	E. P.	21	6/15/46	D-6	Inc.	Right								Yes	Yes	1	Exc.	1 mo.	No	19
34	38	L. S.	21	4/20/45	D-11	Comp.	Right								Yes	No	2	Good	10 days	No	20
35	39	T. S.	20	1/21/45	D-6	Comp.	Left								Yes	Yes	2	Exc.	6½ mo.	No	21
36	40	H. W.	45	7/7/40	D-9	Inc.	Left								Yes	Yes	1	Exc.	3 mo.	No	22
37	41	C. W.	29	4/6/46	D-10	Comp.	Left								Yes	Yes	1	Exc.	3½ mo.	No	23
38	42	J. W.	22	11/14/45	D-4	Comp.	Right								Yes	No	1	Exc.	6 mo.	No	24
39	43	C. Y.	25	12/7/44	D-3	Comp.	Right								Yes	Yes	1	Good	3½ mo.	Yes	25
40	44	K. D.	26	3/28/45	L-1	Comp.	Right								Yes	Yes	1	Exc.	3 mo.	No	26
	45						Left								Yes	Yes	1	Exc.	6 mo.	No	27
41	46	M. W.	28	4/28/45	D-2	Comp.	Right								Yes	Yes	1	Exc.	5 mo.	No	28
	47						Left								Yes	Yes	1	Exc.	4 mo.	Yes	29

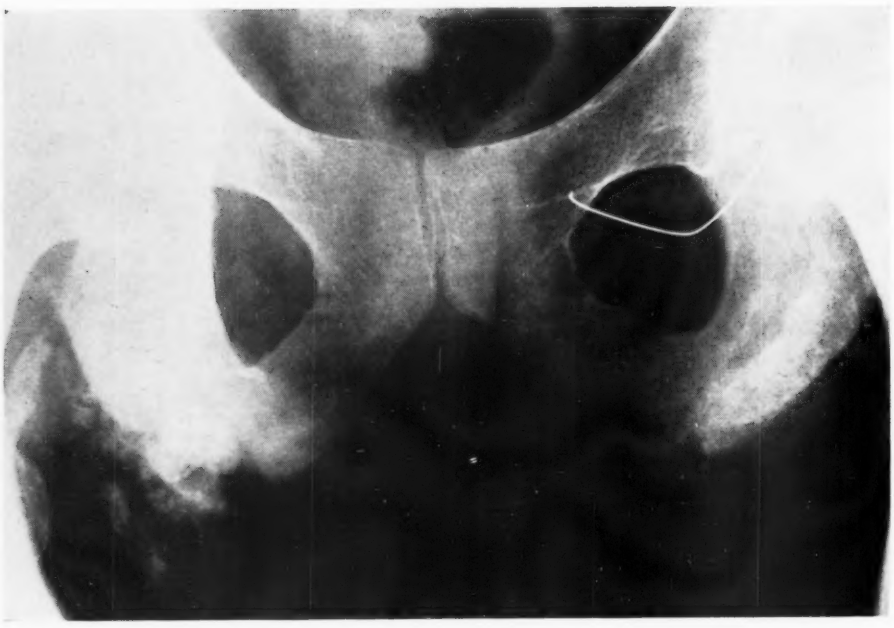


Fig. 2.—Same case as Fig. 1, four weeks after operative repair on the left. Some calcification remains between the muscle planes but ischial contours are smooth. Right side is unchanged.

Fig. 3.—H. M., aged 26 years, level of the fourth dorsal vertebra, date of injury Oct. 6, 1944. Right ischial decubitus ulcer with slight periosteal proliferation and heavy sclerosing osteitis of ischial tuberosity, causing eburnation.

contact on sitting. Whereas the bursa musculi glutaei maximi lies between the muscle and the tuber ossis ischium, the bursa musculi bicipitis superior separates the long head of this muscle from the ischial tuberosity. At times only subcutaneous tissue separates the skin from the periosteum. Trauma with initial ischemia is quickly followed by necrosis and infection. This may spread into preformed bursae, and may even travel along muscle planes to adjacent structures, especially to the ischiorectal fossa.

The poor vascularization of this region with its predominance of fatty tissue accounts for the low degree of resistance. Infection of the periosteum is followed by periostitis and osteitis in the spongious bone structure of the tuberosity (Fig. 3). The proximity of the anus favors continuation of infection. The bacterial flora encountered in this group of cases consisted of *Pseudomonas aeruginosa*, hemolytic *staphylococcus aureus*, beta streptococcus, *Aerobacter aerogenes*, *Escherichia coli*, *Bacillus proteus*, and Catarrhalis. This corresponds with the findings of other authors.[9, 12] The microscopic findings revealed chronic inflammatory nonspecific granulation tissue with hyaline coagulation necrosis and infiltration with round cells with occasional eosinophiles. The picture was essentially dense fibrous tissue and capillary proliferation. Frequently calcific deposits were found which had undergone metaplastic ossification and cartilage formation (Fig. 4, *A* and *B*).

The gross appearance varies. There are ulcers of long standing which reveal only a small outer opening leading into the bursal sac (Fig. 5), while there are others which show a large area with undermined edges and exposed deep structures (Fig. 6). The various stages from the early superficial bruise to the final fully developed ulcer are like those generally observed in sores in any other location.[17] The enumerated anatomic facts and the additional impairment of blood supply by disuse atrophy explain the lack of an efficient defense barrier. They also explain why conservative treatment with penicillin jelly, Furacin, granulated sugar, or cod liver compounds is ineffective, while it yields good results in bone lesions or other decubitus ulcers.[6, 13, 15, 30]

PREOPERATIVE TREATMENT

The preoperative treatment is essentially the same as in ulcers of other locations.[3, 4, 5, 9-13, 17, 18, 20, 31, 32] It is directed toward improvement of the general and local condition. Anemia must be corrected first, especially in the presence of hypoproteinemia, because protein administered in anemia is utilized for restitution of hemoglobin.[20] The protein loss from sores has been found to be as high as 50 mg.[3, 4, 20] A reversed albumin/globulin ratio suggests a depletion in proteins.[20] The importance of a positive protein balance has been stressed.[25, 27] Therefore, amino acids should be administered by mouth and vein in cases of deficiency. But even in the presence of a positive protein balance, and a normal blood count, regular blood transfusions before and after, one to two times per week, starting two weeks prior to intervention and continued for two weeks after operation, have proved very satisfactory. In addition, avitaminosis must be corrected.[3, 4, 5, 9, 11, 12, 18, 31, 32] Insulin was recom-

A.

B.

Fig. 4.—*A*, B. H. S. aged 24 years, level of the fourth dorsal vertebra, date of injury April 2, 1945, left ischial decubitus ulcer. Hemolysin and eosin ×50 showing calcium deposits with metaplastic bone formation. *B*, Hemolysin and eosin ×100 showing neurogenic atrophy of skeletal muscle fibers with increase of sarcolemma nuclei.

Fig. 5.

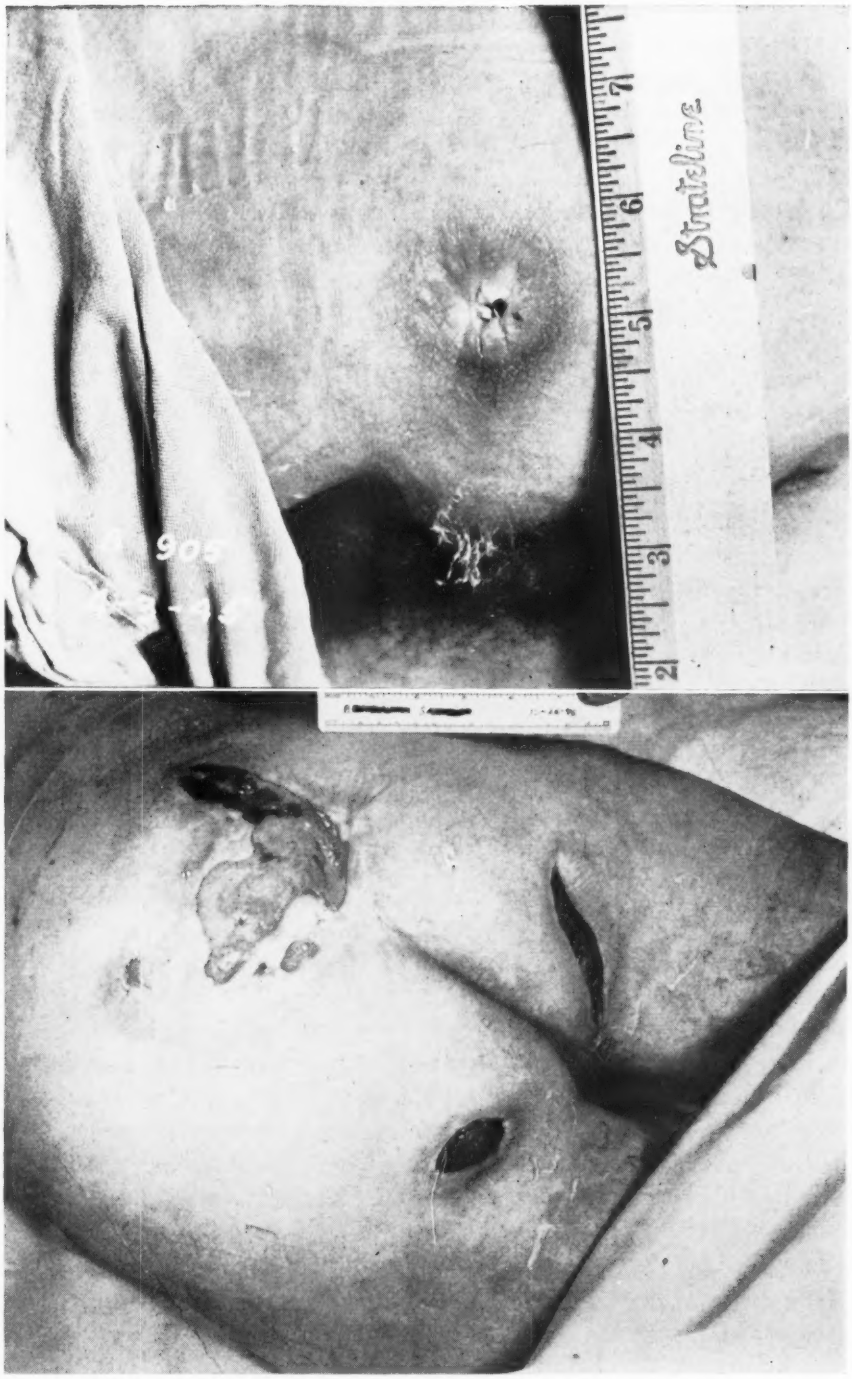


Fig. 6.

Fig. 5.—L. E. S., aged 24 years, level of the fourth dorsal vertebra, date of injury Aug. 29, 1943. Left ischial decubitus ulcer, of 1½ years' duration, small opening, large bursal sac, calcified with osseous involvement.

Fig. 6.—B. H. S., aged 24 years, level of the fourth dorsal vertebra, date of injury April 2, 1945. Bilateral ischial decubitus ulcer with undermined and widely separated skin edges, exposing underlying structures. Large bursal sac with calcification and osseous involvement.

mended in the presence of anorrhexia.[1] Sytemic penicillin and sulfadiazine (where tolerated) are started twenty-four to forty-eight hours prior to surgery.[9, 13, 22, 31, 32]

The local treatment consists of repeated débridement followed by Dakinization through tubes with protective zinc oxide ointment for the skin. Other authors recommend hypertonic saline solution and aluminum acetate,[10] tyrothricin gauze-mesh,[17] or specific local therapy with antiseptics.[9] In the absence of slough good granulations are maintained by wet dressings of Domeboro's solution.* No attempt was made to sterilize the ulcers in concurrence with some[3, 4, 5, 17, 18] and in contrast to other authors[9, 11] who defer operations in presence of certain microorganisms. When no necrosis is present, the granulation tissue appears strong and healthy with marginal epithelization, there is a positive nitrogen balance, and a gain of weight, the patient is ready for surgery.[11, 12]

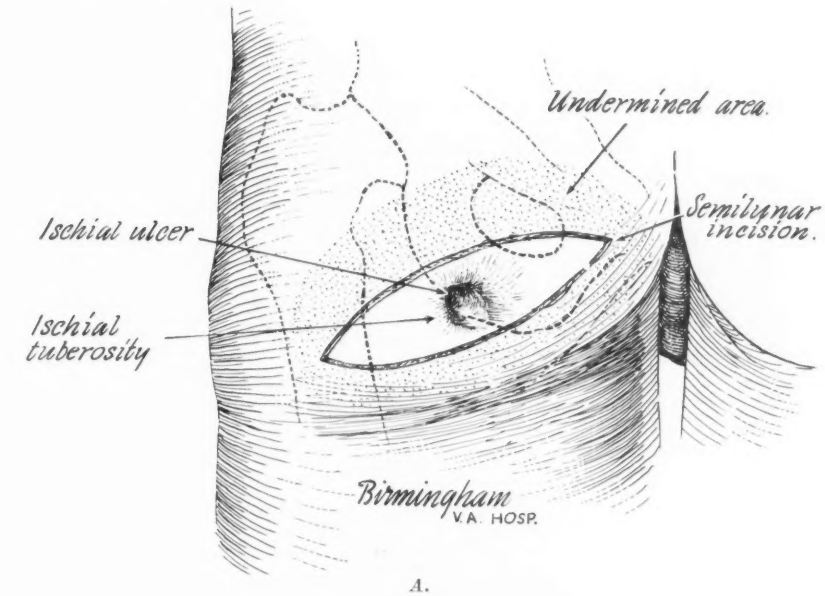
The general postoperative management is a continuation of the preoperative regime. Penicillin and sulfa therapy are carried out for ten to fourteen days. Blood transfusions are administered once or twice a week. The drains are loosened after three to four days and removed around the fifth day. The sutures are removed after ten to twelve days except the retention sutures which are left in place as long as necessary, even up to fourteen days.[17, 32] During the entire postoperative period no weight-bearing is permitted for at least six weeks. Frequent change of position is carried out but no plaster immobilization[12] is used.

SURGICAL TECHNIQUE

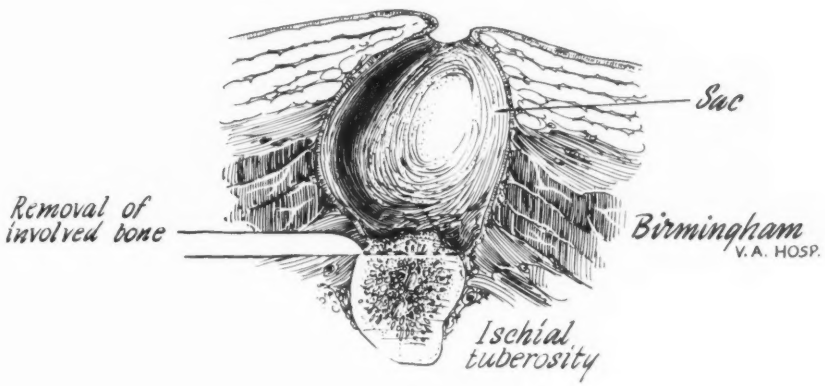
The lesion is excised en bloc from a transversal approach, parallel to the crease. If the bone is not involved, only the bursa is excised and the glutaeus maximus muscle pulled over the tuberosity by interrupted chromic 0 sutures. In cases of bone involvement (primary method) again an attempt is made of removal en bloc, but this includes the periosteum and bone (Figs. 7 and 8). Free use is made of chisel and rongeur. The bone should be smoothed after its healthy parts have been reached. It is covered by muscle in two different ways. Where there is a well-mobilized glutaeus it can be approximated by an inverting stitch of .0126 stainless steel wires on buttons (Fig. 9*A*, primary method). Where muscle is not redundant or when there is a recurrence of the ulcer a mesially pedicled flap of half the thickness of the glutaeus maximus is formed and there is rotated into place to cover the bone and it is attached to the surrounding structures by chromic catgut sutures (Fig. 9*B*, secondary method). Both methods aim at adequate muscle padding of the ischial tuberosity.

Hemostasis is achieved by electrofulguration, hot saline pads, and plain 00 catgut for major vessels. Preferably one or two drains are used. Retention sutures on buttons are used where indicated. Stainless steel .008 and .005 are used for the skin. A fluffy gauze compression dressing is applied with elastoplast, reinforced by an elastic ACE spica bandage.

*The Domeboro solution consists of a mixture of calcium acetate and aluminum sulfate. One tablet dissolved in 8 ounces of water makes a solution of aluminum acetate 0.5/100.0 which is free from lead. One-half of the normal strength was used in the reported cases.



A.



B.

Fig. 7.—*A*, Outline of elliptical skin excision in order to remove the ulcer down to the bone. *B*, Prospective line of removal of bone with fundus of bursal sac.

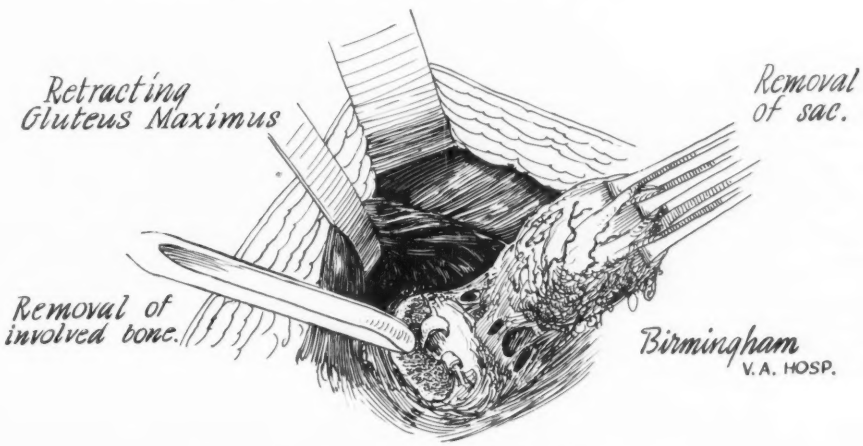
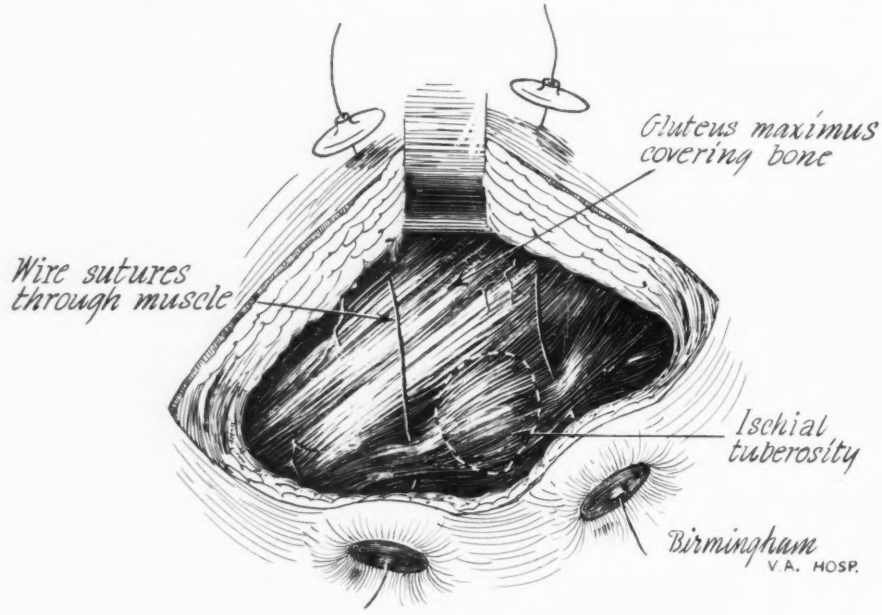


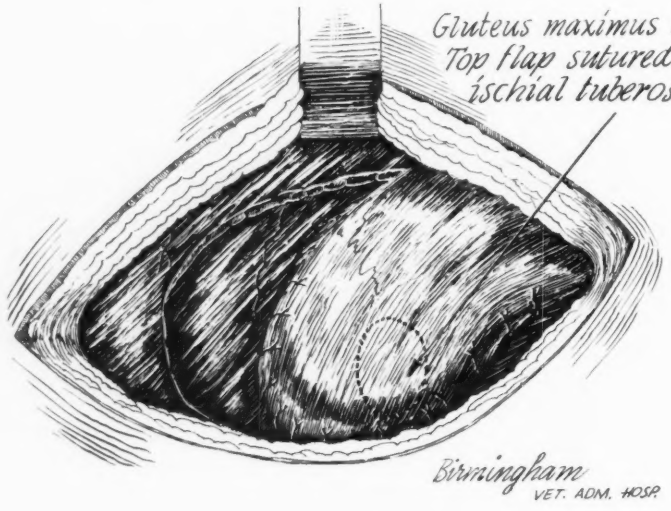
Fig. 8.—Removal of entire sac with adherent bone.

RESULTS

The results are evident from Table II. The total cures with all methods were 85.1 per cent, the total of failures was 15 per cent; 66 per cent responded with healing to one operation, and 19 per cent closed after repeated operations. The breakdown of cures with the simple primary method as compared with the pedicle muscle flap reveals 62.5 per cent against 72.4 per cent, and is in favor of the pedicle muscle flap. If one compares the recurrence rate based upon the



A.



B.

Fig. 9.—*A*, Primary Method: Button and stainless steel wire invert the mobilized muscle and keep it in place in order to cover the ischial tuberosity. *B*, Secondary Method: Muscle flap with medial pedicle half thickness of gluteus muscle rotated in place to cover ischial tuberosity.

number of patients rather than number of ulcers one finds an even more favorable figure of failure in only 15.3 per cent with the pedicle procedure as against 28.5 per cent with the primary method.

The seven failures comprise those ulcers which up to date did not heal. One case was due to faulty interpretation of the x-rays (Fig. 3). The bone changes remained unrecognized and untreated. The second case ended in failure of both methods due to many incidents. The patient fell out of bed, another time became too spastic and was not immobilized enough, and finally a technical error prevented the desired result. Although the sore closed by granulation and buried epidermis graft, the case was considered a failure. Prolonged sitting and a periproctitis produced two recurrences in the third patient, whose ulcer stayed closed for periods of three months. Two patients were uncooperative and sat for periods of more than eight hours driving their cars. The pedicle flap withstood this trauma but the skin broke down. A sixth case failed due to a postoperative hematoma despite drainage and the seventh patient had to go on an emergency leave before a stable scar developed.

TABLE II

	NUMBER OF CASES	PER CENT
Total cures with all methods	40	85.1
Total cures with one operation	31	66.0
Total cures after repeated operations	9	19.0
Total failures	7	15.0
Total	47	100.0

TABLE III. PRIMARY METHOD (21 CASES, 24 ULCERS)

	NUMBER OF ULCERS	PER CENT
Excellent without recurrence	15	62.50
Primary excellent with recurrence	4	16.76
Good without recurrence	1	4.16
Good with recurrence	1	4.16
Poor	3	12.50
	24	100.08

TABLE IV. SECONDARY METHOD (26 CASES, 29 ULCERS; OVERLAPPING IN BOTH METHODS, 6 CASES)

	NUMBER OF ULCERS	PER CENT
Excellent no recurrence	21	72.413
Excellent with recurrence	2	6.996
Good no recurrence	3	10.347
Good with recurrence	1	3.448
Poor	2	6.996
	29	100.200

No relationship between the duration of an ulcer and the outcome of treatment could be established.

The result was called excellent in the presence of primary union, good when a small separation of the suture line occurred (1 to 2 cm.) which closed by delayed healing, and poor where frank nonunion persisted.

DISCUSSION

Freeman[17] mentioned two cases with breakdown after skin flap procedures but did not give the total number of ischial ulcers. White and Hamm[32] re-

ported one case, Croce and Beakes[12] nine cases, four of the patients operated upon with two cures and two failures, and Conway and co-workers[9] 30 cases, fifteen of the patients operated on. Conway and co-workers stated that they had seven cures and eight failures. Our figures therefore compare favorably with those of other authors.

The incidence of ischial ulcers based upon the total number of sores was given as 10 per cent by Conway and co-authors.[9] Our observations concur with their findings. However, if based upon the number of patients our figure would be about 25 per cent. It is to be expected that this ulcer, as a "late" sore will occur even in well-balanced patients at any time. It may recur even after a "sore-free interval" of one year or longer. The patients must abide by their instruction for preventive measures. It also will be difficult to analyze what ought to be called a recurrence due either to inadequate operative procedure or to negligence on the part of the patient.

Skin flap methods have been advocated generally in the literature[3, 4, 5, 9, 17, 18] against linear incisions in the treatment of bedsores. However, it has been stated that a flap can break down[9, 17, 32] and we confirm this experience. We feel that the muscular padding of ischial ulcers establishes a workable compromise between an internal flap and an external linear incision. Ostectomy is essential in the management of ischial ulcers, and where possible this should be done en bloc with the bursa. Conway and co-authors[9] have recently recommended ostectomy in ulcers of different localization.

Different kinds of suture material have been used by various authors. Cotton,[12, 18, 22] tantalum,[13] and stainless steel for tension sutures[32] and ligatures[2] represent the nonabsorbable material. Plain catgut[14] and chromic catgut[4, 5, 9, 31] were used as absorbable material. Silk,[4, 31] horsehair,[31] and waxed silk[14] have been used for the skin. From the results reported it appears that it is not essential what material is used. We like stainless steel[8, 23, 26] with its resistance against body fluids and absence of capillary action. We prefer electrocautery for hemostasis in concurrence with Gibbon and Freeman[18] even though it has recently been rejected.[9] We also agree with the experience[18] that the bacteriologic flora does not play a decisive role. Proper postoperative position and immobilization are important. We have not yet adopted the plaster of Paris spica.[12] Weight-bearing prior to six weeks postoperatively jeopardizes the result.

In order to prevent the development of ischial sores rather than to devise better means of therapy it is necessary to teach the patients how to achieve this goal. Medical instruction to the individual patient and to the paraplegic patients as a group has been attempted. This is the more important because of the psychology of the paraplegic patient which follows its own pattern.[29] The prevention consists of taking off the weight at regular intervals while sitting and the early use of braces.

When an ulcer has developed, its treatment will be indicated along the following lines: a simple ulcer with bursa formation could be treated by excision; a primary ulcer with bone involvement and ample, redundant gluteal muscu-

lature is approached by the primary method. All other conditions, especially recurrences, should be treated by the radical secondary method with a pedicled muscle flap.

SUMMARY

1. Forty-seven ischial ulcers are presented in forty-one patients.

2. The "sore-free" interval from the time of injury to the onset of the ulcer ranged from three months to thirteen years.

3. There were two cervical, sixteen upper dorsal, seventeen lower dorsal, and six lumbar lesions. Secondary bone changes were present in thirty-five cases but no relationship between level of injury and secondary bone changes could be established.

4. Local trauma plays the major role in the etiology of the ischial ulcer. It is believed that the skin circulation is unimpaired but that the neurogenic disuse atrophy of the underlying structures is a contributory factor. This "late" ulcer might develop in the absence of protein disturbances, anemia, or infection.

5. The tendency of the infection to spread to deeper tissues is favored by the presence of poorly vascularized bursae and the proximity of the anus. True metaplastic ossification and cartilage formation is found on histologic examination. The bacterial flora is similar to that encountered in the concurrent urinary infection. Conservative treatment of a fully developed ulcer is unsuccessful.

6. The local preoperative treatment consists in débridement. The general preoperative treatment should correct hypoproteinemia, avitaminosis, and anemia. Multiple blood transfusions are beneficial even in the absence of hypoproteinemia or anemia. Sulfonamides and penicillin are used systemically. Postoperatively a similar regime is carried out as preoperatively. Sutures are removed at a late date.

7. Two methods of surgical treatment are presented. Both remove the ulcer en bloc, including the bone. The "primary method" uses the redundant gluteal musculature for the covering of the bone defect by an inverting stitch of stainless steel wire. The "secondary method" uses a pedicled gluteal flap for the padding of the bone. Stainless steel is used for skin closure. Drainage is important.

8. The total number of cures was 85.1 per cent, the total number of failures, 15 per cent; 66 per cent healed after one intervention, 19 per cent needed more than one operation. The number of cures with one intervention and the number of recurrences is in favor of the "secondary method." The causes of failure were faulty interpretation of x-rays, technical errors, lack of immobilization, and lack of cooperation of the patient.

9. The results presented and the technique are discussed in the light of the recent literature.

10. Early use of braces and medical instruction of the patients are recommended for the prevention of ulcers.

11. A simple ulcer with bursa formation is excised. A primary ulcer with redundant muscle and bone involvement is treated by the primary method, and all other conditions by the secondary method.

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ON THE HISTOLOGY OF SURGICALLY REMOVED SYMPATHETIC GANGLIA

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SINCE the etiology and underlying pathology of disorders such as essential hypertension and peripheral vascular disease are but poorly understood, it is not surprising that specimens removed by sympathectomy have been examined with a great deal of curiosity. Even before sympathectomy became a well-established procedure, pathologists were attracted by the increasing evidence of the important role the autonomic nerves play in health and disease, and histopathologic studies of sympathetic ganglia gained at autopsies were carried out. The interest was divided between the neuronal structures and the interstitial and vascular tissues of the ganglia. Many investigators have shown that ganglia are prone to exhibit a variety of histopathologic findings. They list in one form or another the disorder and coexistent changes, with or without advancing hypotheses.[3, 10, 13, 15, 16] Even authors who lean toward the "post hoc, propter hoc" view and consider the underlying pathology of a case responsible for the histologic changes in the ganglia are constrained to qualify their conclusions.[9, 14]

The main difficulty in judging changes in sympathetic ganglia lies in setting down standards of normalcy by procuring suitable controls. Perfectly healthy persons remain in possession of the sympathetic ganglia, while autopsy material stems from patients with some fatal disease or from victims of accidents, where an exact history, data on blood pressure, etc., is rarely available, and is often improperly fixed for careful histologic purposes.

Another pitfall in evaluating the histology of human sympathetic ganglia is the mistaking of changes due to age for pathologic alterations. Age changes simulate or may be identical with pathologic lesions and in many instances it remains a matter of speculation or definition how to classify a certain "abnormal" picture. It has been established by many investigators,[2, 4, 7, 8, 11, 17] who studied from various angles the changes in the human nervous system due to age, that conspicuous changes begin to appear after the second decade of life and progress into old age.

Critical evaluation of the literature leads to the sobering conclusion that almost every finding in ganglia from patients suffering from a certain disorder can be observed in ganglia of persons not afflicted with the disease in question.

The present studies were not aimed at a statistical analysis of the material at hand. The purpose was to examine carefully the histology of a limited number of operative specimens, that is, ganglia removed to alleviate hypertension

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or peripheral vascular disease. The pictures obtained with a variety of histologic stains were then compared with control material, as set forth in the following two paragraphs. A thorough analysis of a larger number of cases and a more detailed correlation with data in the literature are being planned.

MATERIAL AND METHODS

The specimens gained by twenty-four sympathectomies consisted of sympathetic ganglia, chain, and splanchnic nerves where a thoracolumbar sympathectomy was employed (fourteen cases). Lumbar chain and ganglia removed in ten instances for treatment of peripheral vascular disease form the other part of the material. The age of the patients ranged from 22 to 67 years. Several specimens from hypertensive patients were used.* The material was fixed directly after removal, most in 10 per cent formol, some in acid alcohol, and embedded in paraffin. Bodian's activated protargol, Mallory's aniline blue, Verhoeff's elastic tissue stain, cresylviolet and hematoxylin-eosin were used routinely. Separate blocks of chain and splanchnic nerves were stained with osmic acid. Parts of ganglia were used for frozen sections, 300 to 400 micra thick, to visualize the vascular pattern by means of the benzidine technique of Doherty, Suh, and Alexander.[6]

Material from forty-one autopsies of patients ranging from newborn to 83 years of age, tabulated in Fig. 2 and consisting of celiac and sympathetic chain ganglia, had been collected some years before in the Boston City Hospital for other investigations.[2] This control material† was prepared with similar techniques and it proved to be valuable for comparison, since it contained many specimens from patients who had not suffered from peripheral vascular or hypertensive disease.

OBSERVATIONS

The Vascular Bed.—Thick frozen sections stained with benzidine reveal the vascular pattern characteristic for ganglia. A dense regular network appears in children, Fig. 1, *A*, tortuous channels of varying diameters are found in young individuals, while around the twentieth year the irregularities in diameter assume the shape of spindles, ampullae, and spherical dilatations which become well marked in adults and aging persons (Figs. 1, *B* and 2). In hematoxylin-eosin and aniline blue preparations these conspicuous vascular ampullae measuring between 25 and 100 micra in diameter were found to possess walls similar to that of a capillary or a small vein (Fig. 3), or in many cases a hyalinized layer surrounded the intimal lining. They occur only within the ganglion and are probably responsible for the statements of pathologists who describe engorged vessels, hyperemia, or increased capillary bed,[8, 15] an impression based on examining only stained thin sections. The 300 to 400 micra thick slabs of tissue, however, disclose the vascular tree in three dimensions. The vascular pattern appears to be independent of the underlying pathology and more nearly related to age. Although the size and number of the vascular dilatations do not pro-

*Generously contributed by Dr. R. H. Smithwich, Boston.

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gress strictly with age, no adult ganglion shows the delicate net of vessels found in children. Ganglia from senile persons tend to exhibit a scanty vascular bed.

Within the ganglia one sees many vascular lumina encased in a thick hyaline wall (Figs. 5, 7, and 8). They were found in the majority of patients who had suffered from essential hypertension or peripheral vascular disease. They were absent in all children and young persons, including two patients, 21 and 33 years of age, on whom a lumbar sympathectomy had been performed for peripheral vascular disease. The vessels thus affected do not show an internal elastic membrane; rather, their media consists of a hyaline mass, exceedingly

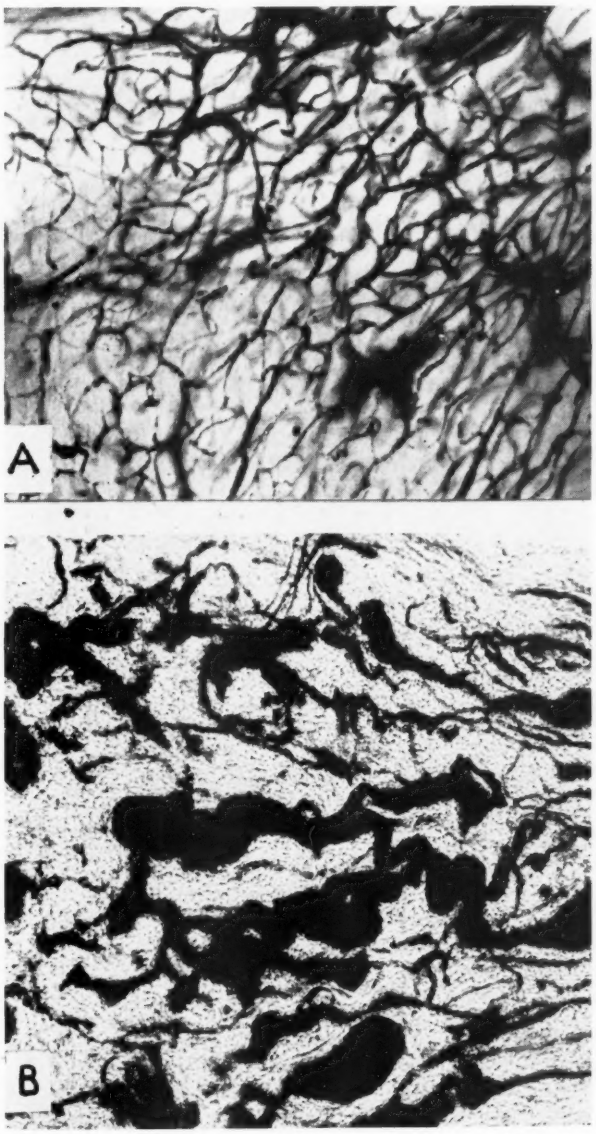

Fig. 1.—*A*, Vessels in a sympathetic ganglion of a 16-month-old male infant (meningococcal sepsis). (Benzidine stain. ×70.) *B*, Vessels in a sympathetic ganglion of a 43-year-old man with rheumatic heart disease. (Benzidine stain. ×60.)

poor in, or entirely devoid of, nuclei. Aniline blue preparations show that these hyaline areas contain loose smokelike strands of reticular fibers (Fig. 8). Transitions from normal capillary or venous walls to fibrous thickenings up to the described hyaline halos do exist. It is of interest that sometimes hyalinization apparently encroaches on the environment of the vessels so that a vessel may appear to lie within an irregularly shaped halo of an almost structureless mass. A portion of the circumference of the vascular wall may remain normal while the remainder exhibits hyaline projections. It is possible to trace some of these vessels into extrinsic veins. The hyalinized tissue then blends with the fibrous capsule of the ganglion. Outside the ganglion the vein retains its normal delicate wall.

Although the arterioles within the ganglia removed at operation may show hyaline walls and/or intimal thickenings, some severe cases of essential hypertension revealed only normal arterioles in the ganglia. In general, arteriolar changes were much less frequent than hyaline halos observed about the capillary or venous channels.

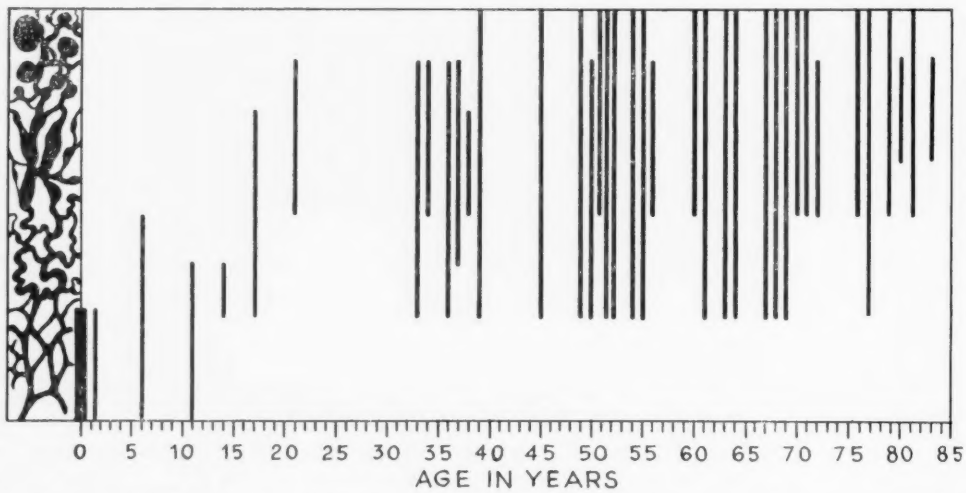

Fig. 2.—Graph showing types of vascular pattern in sympathetic ganglia of forty-one autopsy cases. The thick line at the age 0 indicates four newborn infants.

Infiltrating Processes.—Circumscribed loci of cellular infiltrations are a common finding in the ganglia described in the literature as well as in the material at hand. Round cells or polymorphonuclear leucocytes, or both, are found to form dense perivascular cuffs, larger areas of infiltration covering nerve cells, fibers, and vessels. They sometime lie within the wall of a vessel and can be continuous with intra- or extravascular infiltrations. Control ganglia of children and young persons did not show cellular aggregates, but they were lacking as well in some old individuals, including hypertensives and patients with peripheral vascular disease. In Staemmler's long list,[15] the incidence of inflammatory infiltrations increases with age. In our material a connection between "sympathetic ganglionitis" and clinical diagnosis of the individual case could not be established.

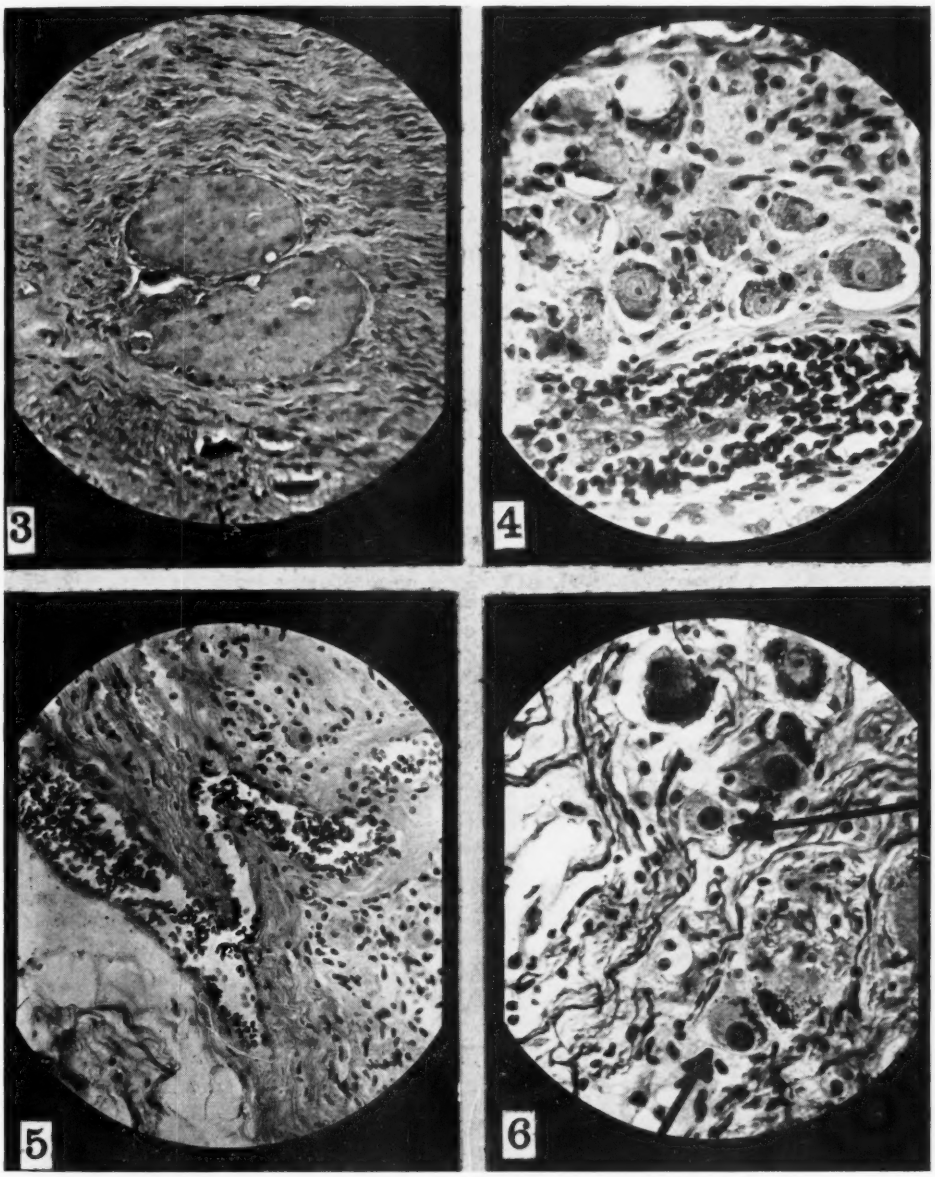

Fig. 3.—Dilated thin-walled vessels from a lumbar sympathetic ganglion of a 22-year-old man with lumbar sympathectomy and peripheral vascular disease. (Verhoeff's stain, ×150.)

Fig. 4.—Infiltration in a lower thoracic chain ganglion of a 47-year-old woman with thoracolumbar sympathectomy and essential hypertension. A vacuolated nerve cell is seen at the top. (Cresylviolet stain, ×250.)

Fig. 5.—Thickened wall of an intrinsic vein in a lumbar chain ganglion, emptying into a normal vein outside the capsule of the ganglion of a 53-year-old man with lumbar sympathectomy and peripheral vascular disease. (Hematoxylin-eosin stain, ×170.)

Fig. 6.—Two small cells without processes from a lumbar chain ganglion of a 66-year-old man with lumbar sympathectomy and peripheral vascular disease. (Bodian's silver method counterstained with acid fuchsin, ×290.)

The Interstitium.—The connective tissue of ganglia consists mainly of its outer collagenous and elastic capsule, septa, and perivascular sheaths. The reticulum and other framework tissue increases in coarseness and density with age. In extreme cases one can observe veritable fibrosis of a ganglion at the expense of neuronal elements. Whether or not this situation is due to inflammatory processes, as claimed by Staemmler,[15] requires verification. Children's ganglia excell in delicacy and sparsity of framework tissue. The sheaths of Key-Retzius and the reticulum around the capillaries are loosely woven, while in adults the framework is denser, coarser, and more extensive. The degree of fibrosis in older persons, however, did not correspond exactly with age, the severity of vascular alteration, or the incidence of infiltrating processes.

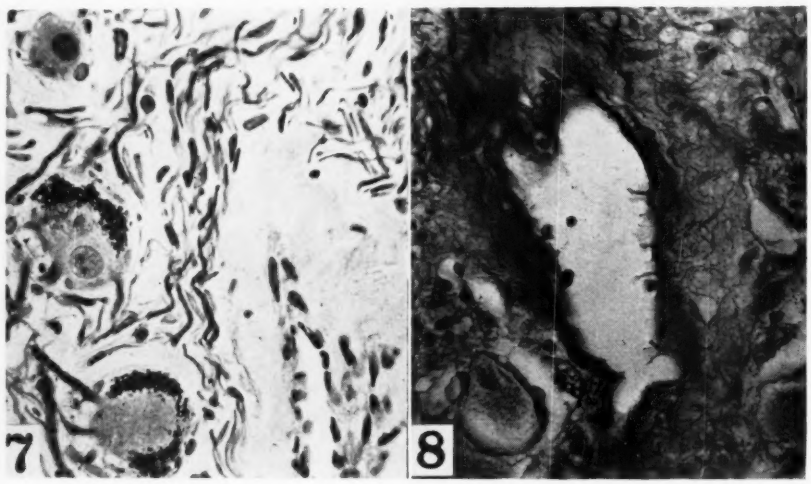

Fig. 7. Fig. 8.

Fig. 7.—Binucleated pigmented ganglion cell and hyalinized vessel from a lumbar chain ganglion of a 57-year-old man with lumbar sympathectomy and peripheral vascular disease. (Bodian's silver method counterstained with acid fuchsin. ×320.)

Fig. 8.—Greatly thickened vascular wall in a lumbar chain ganglion of a 53-year-old man with lumbar sympathectomy and peripheral vascular disease. (Mallory's aniline blue stain, ×320.)

The Neurones.—Cresylviolet stained sections of sympathetic ganglia reveal a great variety of abnormal cells, interspersed among a majority of normal elements. Paucity of Nissl substance, vacuolization of cell bodies, chromatolysis, swollen pale cells and spacious cell capsules containing foamy detritus, pyknosis or absence of nuclei, ghostlike cell shadows, and increase in the number or density of satellite cells are the most common findings. We did not feel justified in following Laignel-Lavastine[13] who considered marginal position of the nucleus and even excentric position of the nucleolus as abnormal. The incidence of cellular changes is exceedingly low in the child. In autopsy and operative material gained from adults, they defy correlation with age and diagnosis. According to Kuntz,[10] and Kuntz and Sulkin,[12] the appearance of the Nissl substance and of the satellite cells changes with the functional state of the cell. It was possible to see in our series alterations they described, but since normal elements

could always be found in the same ganglia and since presumably normal individuals also showed similar pictures, we were reluctant to draw any conclusions. Bodian's silver stain applied to ganglia gives a similar variety of abnormal findings. Heavy loads of argyrophil pigment in neurones of adults, haziness or absence of neurofibrillar structure, thickening of dendrites, and fenestration of nerve cells can be seen. Knotted and beaded axones, indicating degeneration, were observed singly or in strands. The pictures seen correspond to the forms described by De Castro[5] in his account of normal and pathologic sympathetic neurones. Binucleated nerve cells can be found in all age groups (Fig. 7). Their significance is discussed by Beaton, Holmes, and Windle.[1] There is no evidence that they represent pathologic forms. A somewhat aberrant type of cell was met in most of the silver preparations. They were smaller than the average nerve cell, round or polygonal with blunted corners, apparently without processes. The nucleus was denser than that of ordinary neurones, the cytoplasm homogeneous without fibrils, but occasionally laden with argyrophil granules, finer than the pigment in typical nerve cells (Fig. 6).

COMMENTS

The results indicate that the histology of sympathetic ganglia does not give us information about the severity of vascular disease. Although the hyalinized vessels in our series were best developed in some cases with Buerger's disease, differentiation of essential hypertension from peripheral vascular disease is not possible, since hyalinization is a common finding in either one of the two groups of patients. The severity of the disease as judged by duration, blood pressure, and clinical course is not proportionate to the degree and extent of morphologic alterations. It appears rather that the ganglia are not a sensitive gauge for recording the diseases in question, since they suffer to a varying degree under pathologic and aging processes of the entire organism. More light could be shed on the histopathology of sympathetic ganglia by accumulating more control material from aging persons not afflicted with vascular disorders. A critical evaluation of the literature together with our findings leads to the conclusion that there is no specific histologic picture for hypertension or peripheral vascular disease in sympathetic ganglia. This harmonizes with the view that sympathectomy, although a valuable aid in denervating the vascular bed or interrupting harmful visceral reflexes, probably does not constitute an attack against the source of vascular disease.

SUMMARY

1. Ganglia removed by sympathectomy and stained by a variety of techniques have been compared to routine autopsy material, of known age and diagnosis.

2. Abnormal findings in sympathetic ganglia of adults are exceedingly common and include: degenerating neurones, inflammatory infiltrations, increase of interstitial tissue, and hyalinized thickened walls of intrinsic veins.

3. Ganglia of infants and children possess little connective tissue, delicate vascular walls, and few or no degenerate neurones.

4. The most consistent pathologic findings are hyalinized walls of intrinsic channels, usually venules, in most cases of essential hypertension and peripheral vascular disease. However, the incidence and intensity of these alterations are not strictly related to the severity of the disease nor to the age of the patient. Arterioles within the ganglia may present a normal appearance in cases of hypertension or peripheral vascular disease.

5. The findings do not permit the establishment of a histologic picture characteristic for either hypertension or peripheral vascular disease.

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INTERNAL FIXATION OF HIP FRACTURES WITH A NEW LAG SCREW

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FRACTURE of the neck of the femur is the most common accident occurring in later life and is one of the most serious injuries that befall the elderly. Until as late as 1925, when Smith-Petersen presented the three-flanged nail and thus gave impetus to the real development of modern fixation methods, the great majority of these fractures terminated finally in death. The truth of this statement is attested by the fact that in most counties in this country a death occurring within one year following hip fracture must be reported to the coroner of the county in which the death occurred.

The history of treatment of fractures of the hip comprises a voluminous literature, and through it all the method of internal fixation has become an approved and accepted treatment with a large percentage of excellent results and a gratifying drop in mortality rates. Particularly in intracapsular types of fracture has internal fixation proved its value and prevented distressing nonunion.

The lag screw has long been used by mechanics to approximate surfaces when it is impossible or inadvisable to use a bolt. The application of the principle of the wide flanged thread of the lag screw to bone surgery is particularly logical in that it serves to hold the fractured ends more firmly together than any other fixation medium. This was recognized many years ago by Johannson when he brought forth his bone screw, and later by many other surgeons. I have used the lag screw herein described for more than six years and have not found a single instance in which the screw has slipped, or rotation has occurred. On the contrary, when reduction of the fracture is accurate the two fragments are held in such firm apposition that rotation is not possible. In several cases where bone rarefaction and absorption occurred I found the lag screw still held the fragments in exact position, eventually resulting in good union in good position. No force is required in the insertion of the lag screw; therefore, no impaction occurs, and there is no danger of splitting the shaft when going through the hard cortical bone of elderly patients.

The lag screw herein presented fulfills the criteria of rigid fixation as laid down by Watson-Jones a number of years ago, namely:

1. Rotation of fragments should be prevented.
2. Length of splint should be sufficient to prevent angulatory movement.
3. There should be a minimum of bone displacement.
4. There should be a maximum of bone surface contact.

This lag screw is constructed of nonelectrolytic, nonmagnetic molybdenum stainless steel in varying lengths to meet the variation in size of femurs. The

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principle of drawing the two fragments together requires that all of the threaded portion of the screw should be in the proximal fragment. After careful study and measurement of a large number of hip fractures I found that the proximal fragment was invariably a minimum of one inch in length. Because of this constant finding the threaded portion of the screw is less than one inch in length, which suffices for all hip fractures regardless of where the line of fracture runs. The head of the screw is beveled at 130 degrees, which is the average approximate angle of the femoral neck to the shaft. This permits the undersurface of the screw head to lie flat against the shaft instead of digging sharply into the bone. Instead of the conventional slot for the insertion of a screw driver a hexagonal shaft is countersunk in the head into which the hexagonal bar of a special screw driver as illustrated, or that of an ordinary Allen wrench fits securely. This gives complete control of direction and leverage without danger of slipping or sliding of the screw driver.

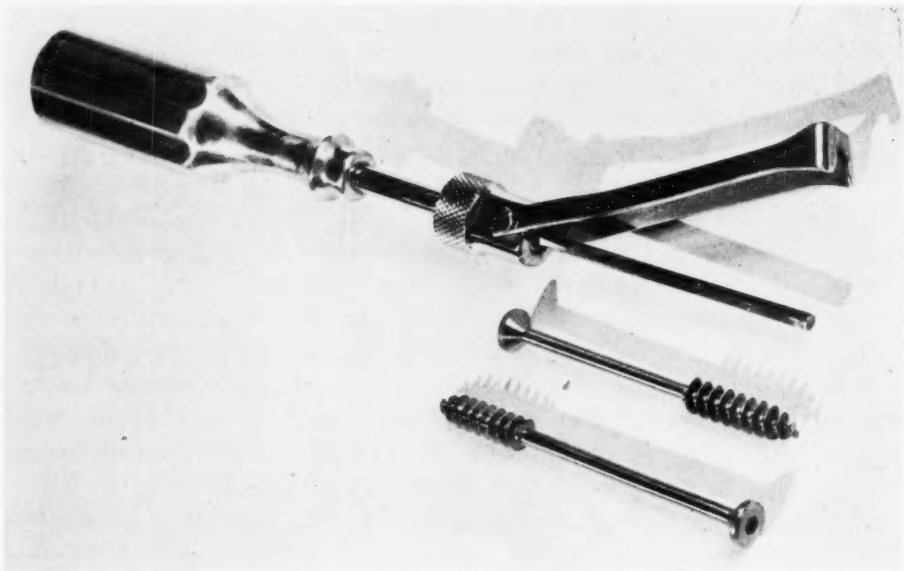


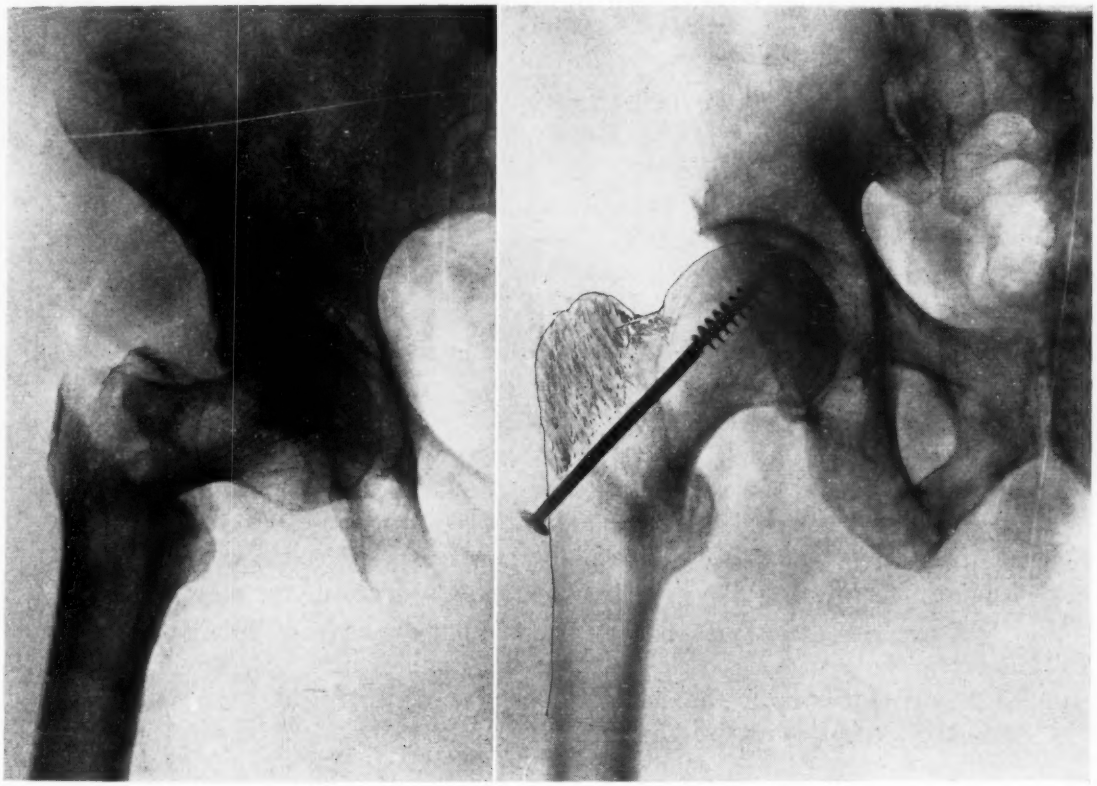
Fig. 1.—Illustration of lag screw herein described, with type of screw driver used. This lag screw is made of molybdenum stainless steel, which is nonmagnetic and nonelectrolytic.

The time to operate is always a question of individual judgment depending entirely upon the condition of the patient, the type of fracture, and the presence or absence of complications. There is usually no need for haste but I have found it most satisfactory to operate within the first twenty-four hours if conditions warrant. In the presence of shock, internal injuries, or complications, it is sometimes advisable to delay fixation for one or two weeks. During this interim the patient should be kept as comfortable as possible, with sandbags or their equivalent holding the injured leg, or in some cases light traction. Anesthesia is purely a matter of choice, although spinal or caudal procaine or intravenous Pentothal sodium is advisable if the operation is to be performed on the x-ray table.

TECHNIQUE

The patient is brought to the operating room following the usual preoperative medication and is placed on an ordinary operating table. A cassette holder replaces a part of the mattress pad to facilitate the taking of films during the progress of the operation, but no other special equipment is needed.

The patient is then anesthetized and reduction of the fracture accomplished by the Leadbetter method or any other successful maneuver. I have personally used the Leadbetter method and have had no difficulty in reducing all fractures. The thigh is flexed to 90 degrees with the knee also flexed to 90 degrees. A folded towel is passed around the upper thigh and traction is applied by an



A. *B.*

Fig. 2.—*A*, Intertrochanteric fracture of right femur; *B*, fracture reduced and fixation with lag screw accomplished.

assistant holding this towel while manual traction is also applied in the axis of the flexed thigh. With traction being applied in these two directions the thigh is internally rotated and the leg is slowly circumducted into abduction and straightened out, the internal rotation being maintained. As the leg is brought down to table level the foot is just over the lateral edge of the table, and the heel-palm test is applied to confirm reduction. A nurse or orderly then holds the leg in this position throughout the operation and x-ray views are taken in

both anteroposterior and lateral planes to ascertain that the fracture has been reduced.

The technique of taking satisfactory roentgenograms in both planes is relatively simple. The cassette holder on the operating table is adjusted to hold a cassette under the pelvis, and a loaded cassette is inserted. For the anteroposterior view the tube is centered over the neck of the femur using the following landmarks: In draping the patient a towel clip is clipped through the sheets and into the skin at a point midway between the symphysis pubis and the anterosuperior spine. With this clip as a guide the x-ray tube is centered over a point one and one-half inches lateral and distal to the clip. In taking the lateral view the tube arm of the portable x-ray machine is placed at a level with the top of the table on the opposite side of the table from the fractured hip at the level of the knees. The well leg is then raised and held with the knee bent, and the tube is focused obliquely at the fractured hip. A loaded cassette is then dropped into a sterile pillow slip held by the surgeon or an assistant, and the cassette is then held firmly against the body just under the costal margin with one edge of the cassette resting on the table, and facing the hip joint at an angle of about 45 degrees to the long axis of the patient. In this position the x-ray film is exposed and gives an excellent lateral view of the head and neck of the femur.

A short incision is made through the skin and subcutaneous tissue in the lateral surface of the thigh, extending downward from a point just below the greater trochanter. The fascia lata is then incised and the muscle is separated down to the femoral shaft. A calibrated guide pin or a long drill is then drilled through the lateral cortex in a line directed to the towel-clip marker previously described. The distance drilled is estimated from measurement of the head and neck of the femur, remembering the distorsion caused by the distance of the femur from the x-ray film as well as the focal distance of the tube.

X-ray views are then taken in both anteroposterior and lateral planes to ascertain the correctness of the position of the pin or drill. If correct, the pin or drill is removed and the length of screw required is more accurately determined by measuring the length of the pin that had penetrated the bone and then figuring from the x-ray picture any additional length needed. A starting tap having the same thread as the screw is then used through the cortex where the pin or drill was inserted, in order to permit the wide threads of the lag screw to traverse the cortex without drilling a hole larger than the shaft of the screw. The lag screw is then inserted and screwed into place, in the direction found correct by the x-ray films when the guide pin was inserted. As the threaded portion of lag screw passes from the soft cancellous bone into the firmer bone structure near the cortex of the head of the femur the screw can be felt to grip into the bone as the fragments are thus drawn snugly together. With the lag screw in place another set of roentgenograms are taken, again in both anteroposterior and lateral planes, and while waiting for the report the incision is closed in layers. Should the x-ray view by chance reveal a misdirection of

the screw it is only a matter of seconds to remove sutures already placed, and time is saved if the picture reveals satisfactory placement of the screw.

The entire procedure seldom requires more than thirty to forty-five minutes, and the patient suffers little or no shock. The patient is returned to bed and the leg placed on pillows with the knee slightly flexed and the thigh moderately abducted. No splint, sandbag, or other immobilizing equipment is necessary. Routine care is given and high back rest permitted as soon as tolerated. The patient is completely ambulatory in bed, and permitted up in a chair in two or three days. Walking with crutches is encouraged as soon as the patient is strong enough to use them, putting the affected leg down on the floor while walking but cautioned not to put any appreciable weight on it for two months. I have had several cases, however, where this advice was disregarded and walking without any crutches was resumed within a few weeks of the fixation, and with no ill effects.

Postoperatively these patients are quite comfortable and have very little pain. They are able to eat and sleep, sit up on back rest, lie on the well side, and move around in bed. In the presence of a complicating arthritis they exhibit considerable pain, but this is probably due to the fracture trauma and the manipulations of the fracture reduction rather than the fixation. Pain continuing more than two or three days should be carefully checked for complications, such as arthritis, or destruction of the blood supply of the proximal fragment with swelling and beginning necrosis.

CONCLUSIONS

I have found this lag screw equally efficacious in intertrochanteric as well as intracapsular types of fracture, utilizing the mechanical principle for which it was originally designed. In adapting it to the fixation of hip fractures, the fact that simplicity of insertion, security in the mechanical immobility of fragments, and a practical one-piece screw that can be used by any surgeon without extensive equipment justifies its use and demonstrates its efficiency.

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EPIDERMOID CYST OF THE SPLEEN

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THE recent observation of an epidermoid cyst of the spleen has prompted us to report the case and to consider briefly nonparasitic cysts of the spleen in general.

In 1829, Andral[1] recorded the first case, presumably a dermoid. From then through June, 1946, there have been reported 163 cases.[2] In a search of the available literature, 15 (9.2 per cent) of the 163 cases have been reported as epidermoid.

The etiology of nonparasitic cysts of the spleen is not known. Various theories have been offered, including those of metaplasia of endothelium[3, 4]; herniation of splenic tissue[5]; pre-existing lymphangiomas[6]; infarction or hemorrhage associated with pregnancy[7]; menstruation, trauma, malaria, syphilis, tuberculosis, rupture of an intrasplenic artery, or infarction due to arterial degeneration[8]; misplacement of the Wolffian body[9]; and of autochthonous formation.[10] We can add nothing concerning their etiology.

Of several classifications of splenic cysts proposed, that of McClure and Altemeier,[11] modified from those of Moynihan[12] and of Fowler,[6, 7] is the most comprehensive, as follow:

I. True Cysts
   - A. Epithelial
     1. Dermoid
     2. Epidermoid
   - B. Endothelial
     1. Lymphangioma
     2. Hemangioma
     3. Polycystic disease
     4. Some serous cysts
   - C. Parasitic
     1. Hydatid - echinococcus

II. False Cysts
   - A. Hemorrhagic
   - B. Serous
   - C. Inflammatory
     1. Acute necrosis in infection
     2. Chronic tuberculosis
   - D. Degenerative liquefaction of infarcted areas caused by embolism or arterial thrombosis.

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The symptoms and signs of nonparasitic splenic cyst vary depending on the size of the tumor and on the presence or absence of adhesions. Roentgenographic studies may be of diagnostic value. Because of the lack of any characteristic clinical picture and of the rarity of the condition, the preoperative diagnosis has been made very seldom.

Fig. 1.

Fig. 2.

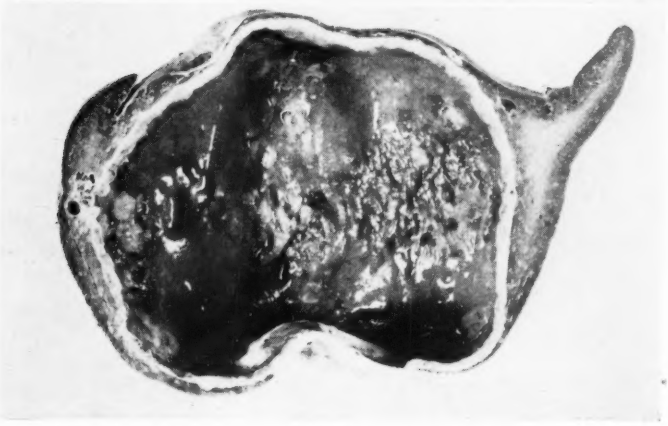


Fig. 1.—The superior surface of the spleen after its removal.

Fig. 2.—The cross section of the spleen showing the enormous unilocular cyst occupying the major portion of the organ, and a small daughter cyst at the extreme left. The resemblance to a gourd of this and other published photographs of similar cysts is striking.

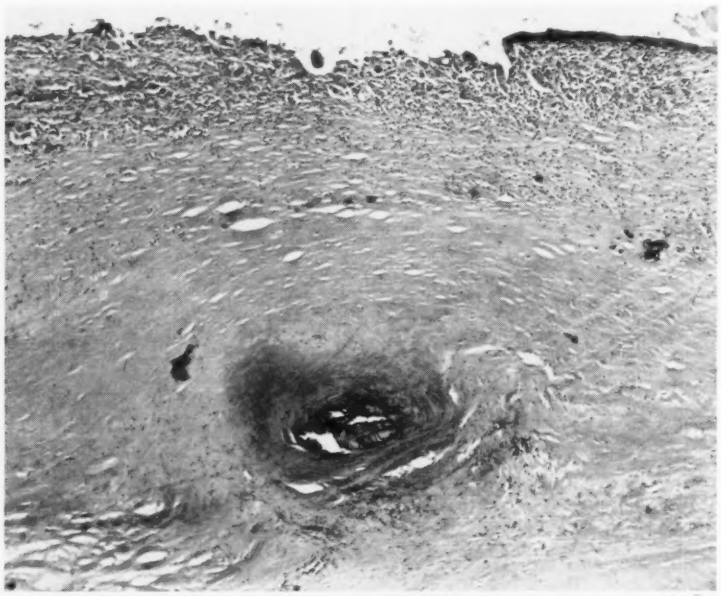
The treatment of choice is splenectomy. This has been accomplished with a low mortality (4 per cent).[13] In those patients surviving, no late complications have been recorded.

Our case of epidermoid cyst of the spleen appears similar to the cases of Pohle,[14] Schneider,[15] Dinand (Lubarsch),[16] Santy,[9] Shawan,[10] Harding,[17] Weil, Scemama, and Roux-Berger,[18] Gosset, Bertrand, and Gosset,[19] Lereboullet and

associates[20] and to the first case reported by Montgomery, McEnery, and Frank.[21] In addition, Custer[22] reported five cases in a series of 5000 autopsies.

A description of the patient observed by us, treated successfully by splenectomy, is recorded here.

Fig. 3.



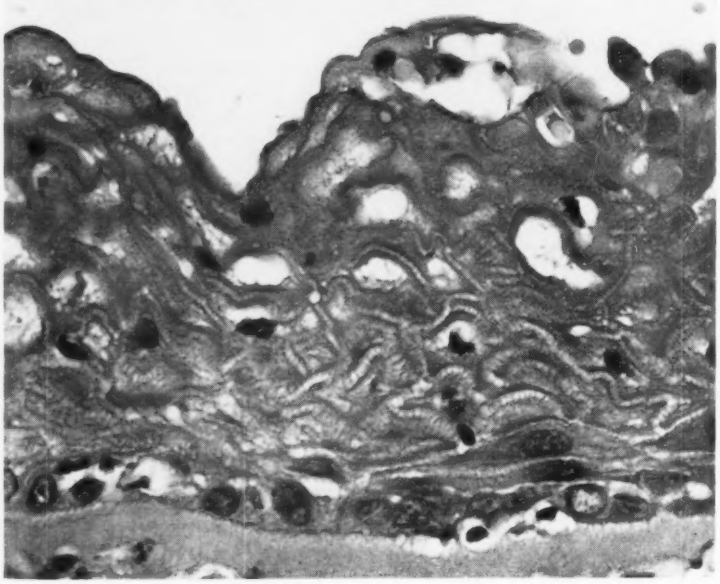


Fig. 4.

Fig. 3.—The photomicrograph (×50) of the wall of the large cyst shows dense collagenous fibrous tissue with foci of calcification, and with some erosion of its epithelial lining.

Fig. 4.—A photomicrograph (×550) of the lining of the large cyst shows stratified squamous epithelium with prickle cells.

Case Report (Roper Hospital No. 48034).—E. F., a Negro female, aged 16 years, was admitted for the third time on Aug. 13, 1946, because of manic depressive psychosis.

A very large mass was found in the left upper quadrant of the abdomen. Its duration could not be ascertained. On the first admission, May 24, 1946, while pregnant, she complained of epigastric pain and vomiting. No specific cause was found. Recovery was spontaneous. No mass other than the large uterus was noted. In an x-ray view of the abdomen the spleen was not outlined. On the second admission, June 18, 1946, she gave birth to a premature male infant, her first child. The puerperium was uneventful. The presence or absence of an abdominal mass other than the uterus was not recorded. After her return home, the patient "went crazy," hence the third admission.

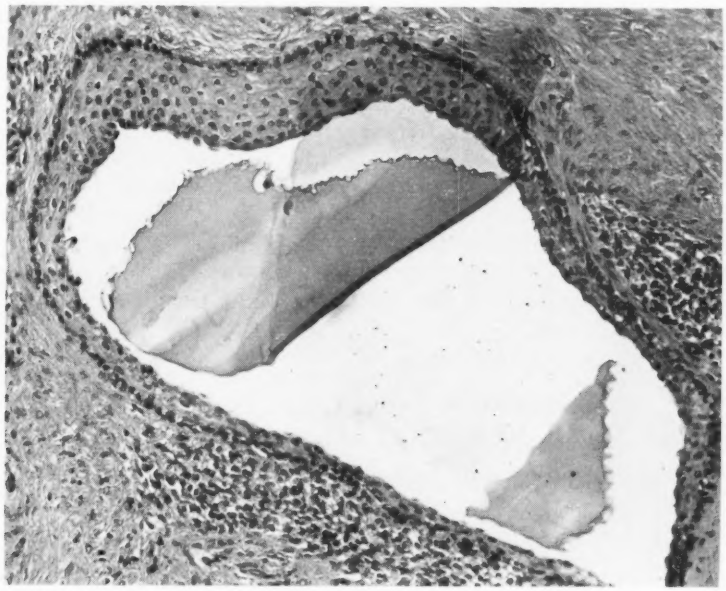


Fig. 5.—A photomicrograph (×150) of the small daughter cyst shows a capsule and lining similar to that of the large cyst.

On physical examination, temperature, pulse, and respirations were normal. Blood pressure was 110/75. There were no abnormal findings except on mental and abdominal examination. She was mentally deranged. The abdomen was asymmetrical. A large bulging mass presented in left upper quadrant, extending three fingerbreadths below the costal margin. It was smooth, firm, freely movable, and nontender. No notch was palpable. It projected from beneath the left costal margin. Its shape was consistant with that of an enlarged spleen. The remainder of the abdomen was normal.

Accessory examinations included the following: red blood cells, 3,900,000; hemoglobin, 10 Gm.; white blood cells 5,200. Differential, hematocrit values, bleeding and clotting times, fragility test, platelet counts, sternal bone marrow, sickle-cell preparations, sedimentation rate, and icterus index were all normal. Blood smears for malaria were negative. Urine and stool examinations were normal. Blood Wassermann and Kline tests were negative. Intravenous pyelograms and gastrointestinal series were normal except for downward displacement of the left kidney and medial displacement of the stomach.

Laparotomy was advised because of splenomegaly of unknown etiology. Malaria was considered the most likely diagnosis.

On Sept. 7, 1946, under ether anesthesia, laparotomy was performed through a left upper paramedian incision. The peritoneal cavity was free of adhesions. Immediately apparent was a large fluctuant spleen measuring 30 by 15 by 15 cm. in diameter. There were

no adhesions about it. Most of the presenting surface was discolored white, within which area there were patches which appeared gray and seminecrotic. Because of its size, it was impractical to remove it intact. Thick, chocolate-colored odorless fluid, 2,000 c.c., was aspirated. The fluid contained many short fine hairs, and small shimmering crystal-like bodies resembling cholesterol crystals. Following this, the spleen was removed after ligation of its pedicle with silk ligatures. All other abdominal organs appeared normal. The wound was closed in layers, without drainage, with interrupted fine silk sutures. Immediately before and during operation, 1,500 c.c. of citrated blood were given although there was no significant blood loss.

Bacteriologic and parasitologic studies on the fluid were negative.

On gross pathologic examination, the spleen was found to be soft and flabby (Fig. 1). On section, it was largely occupied by a cystic cavity measuring 13 by 10 by 11 cm. (Fig. 2), still containing 600 to 800 c.c. of similar fluid. Adjacent to this was a small multilocular cyst 4 cm. in diameter. The wall of the large cyst was 4 mm. in thickness, tough and fibrous. At one end of the cyst there was a small amount of grossly normal splenic tissue. On microscopic examination, the cyst wall (Fig. 3) was thick and collagenous fibrous, showing variable numbers of inflammatory cells, foci of calcification, and many macrophages. The lining of the cyst was well preserved in many areas, and consisted of stratified squamous epithelium showing keratinization and prickle-cell formation (Fig. 4). Sections of the small cyst (Fig. 5) showed, in addition, a well-defined basal layer of its epithelial lining. No hair follicles or other skin appendages could be found in numerous sections.

There were no postoperative complications. The wound healed per primam. Her mental state showed no change. She was discharged from the hospital on Sept. 19, 1946. On Feb. 17, 1947, a follow-up note from a mental hospital reported no later complications.

Comment.—At operation, it was felt that the cyst was a dermoid because hairs were present in the fluid. However, lacking microscopic proof, the cyst is classified as epidermoid rather than dermoid.

SUMMARY

1. A brief review of the etiology, pathology, symptoms, and signs of nonparasitic cysts of spleen is presented.

2. Among 163 cases of nonparasitic cyst of the spleen previously reported, 15 cases (9.2 per cent) have been classified as epidermoid.

3. An additional case of an epidermoid cyst of the spleen, treated successfully by splenectomy, is reported.

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SCIATICA CAUSED BY CYST FORMATION IN OLD HEMATOMA

Report on Three Patients Treated Surgically

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AMONG the patients who have consulted me because of severe back pain owing to fascial fat hernias[1-3] were three women who had, in addition, unusual manifestations of sciatica which could not be attributed to the fascial fat herniations causing the more generalized back pain. In all three, the pain radiated down the leg, and was more severe when the patient was sitting; one patient (Case 3) also complained of discomfort on walking, and displayed an abnormal gait. The sciatic symptoms in all these cases were apparently initiated by trauma, and in all three, removal of a fibrous cyst in the region of the sciatic nerve relieved the pain.

In view of the striking similarity in the history, symptoms, and physical findings in these cases—especially since all of the patients had fascial fat hernias as well as the fibrous cysts involving the sciatic nerve—it would seem of interest to report them as a group.

CASE REPORTS

Case 1.—A woman, aged 46 years, had suffered for ten years from severe backache, which was more intense at night. She had noted a trigger point of pain in the back for years, but the pain did not radiate to the legs. She consulted many different physicians, who treated her for "arthritis," but she obtained no relief. One year before I examined her, she had had a second severe fall, which greatly aggravated the symptoms. About five months before, she began to notice pain on sitting; this was referred to the buttock, and down the left thigh and leg, and had increased steadily in severity. At the time this pain was first noted, the patient had also palpated a soft tumor in the buttock. She had continued to seek relief from the suffering, but all treatment had been ineffective. At the time she consulted me, she had been advised to see a psychiatrist, since her physician had come to the conclusion that the difficulty was psychogenic.

Physical examination elicited tenderness over the fourth and fifth lumbar vertebras and a trigger point of pain in the left sacroiliac region, which was relieved by repeated injections of anesthetic solutions. There was also a large palpable mass in the left buttock. This area was also injected with anesthetic solution. The first attempt to relieve the pain in this way was not successful, but with deeper injections of larger amounts of anesthetic solution, the patient obtained temporary relief from the pain in the leg. The preoperative diagnosis was fascial fat hernia in the left sacroiliac region and lipoma of the left buttock.

At operation, herniated fat was excised over the left sacroiliac region, and a large mass of dense fibrous tissue was widely excised through an incision over the left buttock.

The patient experienced relief both from the back pain and the pain down the leg, after operation. She was examined six months later, and was still entirely symptom-free.

Pathologist's Report.—Sections of the herniated fat showed mature homogenous fat, supported by strands of delicate fibrous connective tissue. Sections of the tumor removed from the buttock showed a cyst with the wall composed of sparsely cellular, densely collagenous connective tissue, richly vascularized with capillaries and arterioles. Focal edema and degenerative change, with swelling and pseudocystic transformation, were present in several areas, along with perivascular exudation of lymphocytes. The inner aspect of the cyst was

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generally devoid of lining except focally for a pseudoepithelial covering, apparently consisting of mesenchymal cyst. Attached to the inner aspect were strands of fibrin and small fragments of blood clot (Fig. 1).

The pathologist's diagnosis was adipose tissue and fibrous cyst, the seat of chronic inflammation.

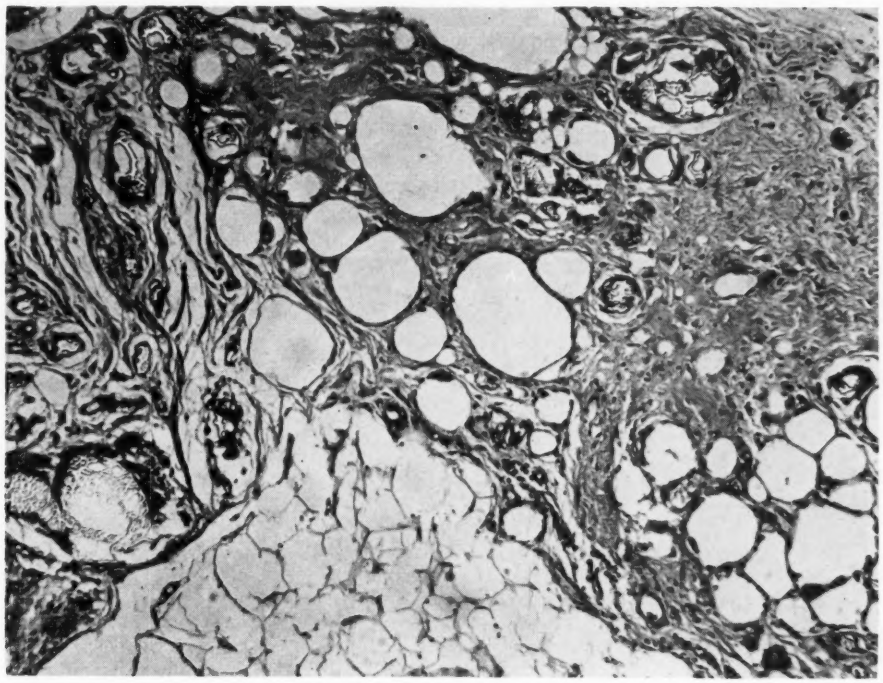

Fig. 1 (Case 1).—Collagenous connective tissue and fat richly vascularized with capillaries and infiltrated with lymphocytes and polymorphonuclear leucocytes. Some of the fat cells are isolated. Giant cells are present in several places.

CASE 2.—A woman, aged 35 years, who was examined in February, 1947, had suffered an injury to the back five years before. She had had continuing pain and had the typical history of having sought relief from various physicians, including orthopedists, and from chiropractors. The pain in the left sde of the back was more severe when the patent was sitting, and radiated centrally down the thigh and leg. The pain also was aggravated with movements of the leg, especially on abduction of the left leg and on flexion of the right thigh on the body.

Physical examination revealed tenderness over the left sacroiliac joint. Five palpable nodules and trigger points of pain were located in this area, and relieved by injections of anesthetic solutions. Deep palpation also revealed a mass in the left buttock, which was also relieved by injection of anesthetic solution. The preoperative diagnosis was multiple herniations of fascial fat.

At operation, several fascial fat hernias were excised from the left sacroiliac region. After closure, another incision was made over the left buttock. Herniated fat was removed and also a cyst, measuring 8 by 10 cm., which was filled with fluid.

The pathologist's diagnosis was adipose tissue, the seat of edema, hemorrhage, and subacute inflammation.

Operation relieved the pain in the back and leg, and five months afterward the patient was examined and found to be symptom-free.

CASE 3.—A woman, aged 46 years, had had a severe fall, landing on her back, three years before I first examined her. Several subfascial fat herniations were found and excised, but this operation afforded little relief. The back remained tender, and the patient complained of difficulty on walking, and of more severe pain when sitting.

Physical examination showed marked tenderness over the cervical, upper thoracic, middle lumbar, and sacral regions. The patient complained that the pain was accentuated when she was sitting. She walked with the right foot everted. In view of the similarity of symptoms in this and the two preceding cases, an attempt was made to palpate a mass in the buttock, but none was found. Nevertheless, injection of anesthetic solution in the right sciatic region afforded some relief from the pain in the leg.

A neurologist who saw her in consultation suggested exploration of the right sciatic region. This was done, with removal of a cyst, 6 by 14 cm., from this area. When the thigh was flexed, during the exploration, fluid was forced out from the lower end of the cyst.

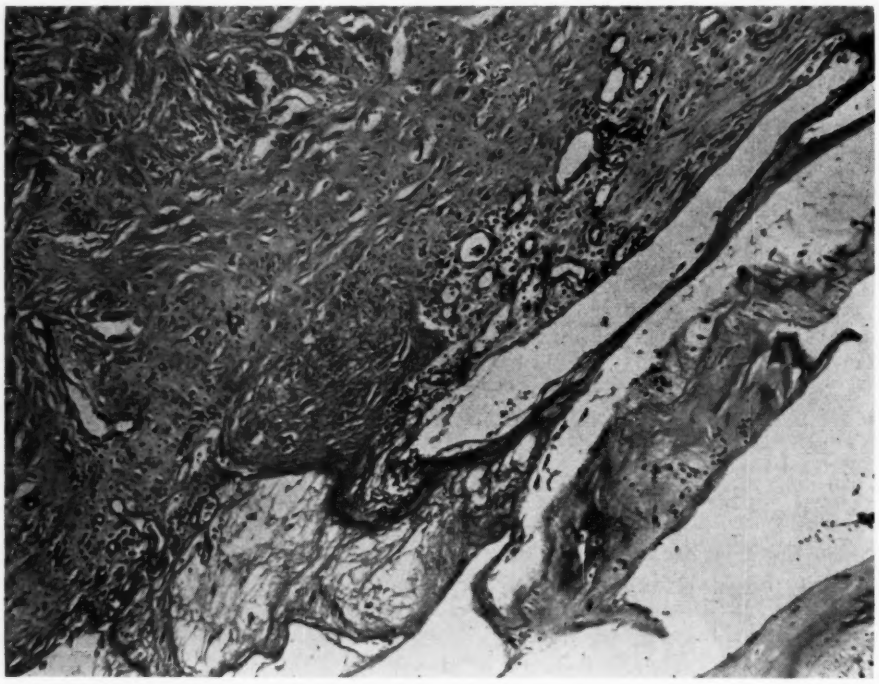

Fig. 2 (Case 3).—Cyst wall composed of dense fibrous tissue and granulation tissue. The latter occurs adjacent to the inner aspect which shows adherent masses of fibrin and blood clot. There is a diffuse lymphocytic exudate.

Microscopic examination showed a fibrous cyst and fibroadipose tissue, the seat of chronic inflammation (Fig. 2).

When the patient was examined three months after operation, she had no discomfort on walking, and the gait was normal. The pain in the thigh and leg had disappeared.

Sciatica, a term first used by Cotugno in 1764, has been applied to any condition characterized by pain in the lower part of the back and the lower extremities, regardless of cause or varying clinical manifestations. It is a general term which has included neuralgia, neuralgia paresthetica, neuritis, radiculitis, protruded intervertebral disk, myalgia, and myositis.

The spinal cord terminates opposite the first lumbar vertebra. The nerve roots forming the cauda equina arise from the lumbar and sacral regions of the spinal cord, descend in the subarachnoid space intradurally, and are freely movable, except as they approach their exits. At the level of the sciatic notch, all these components unite to form one band—the sciatic nerve. The nerve passes around the ischial spine and descends in the posterior portion of the buttock between the ischium and greater trochanter. The pyriform muscle bridges the widest portion of the nerve and occasionally is split by the components of the nerve. The gluteus maximus covers the nerve below this point. The nerve then descends in the posterior part of the thigh into the interspace between the semitendinosus and semimembranosus medially, and the biceps femoris laterally.

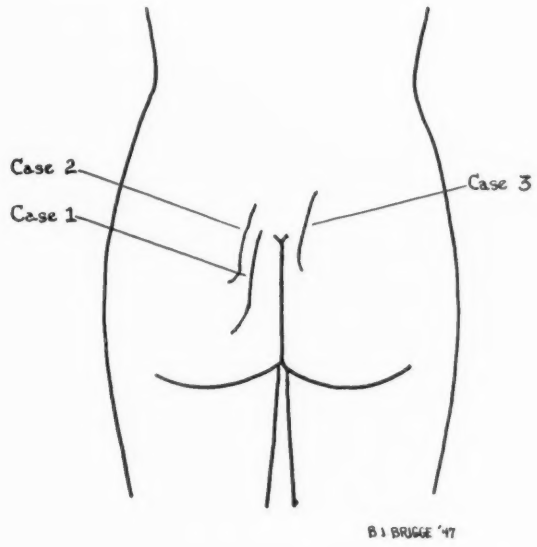

Fig. 3.—Diagram showing situations of cystic lesions causing sciatic symptoms.

Pathologic changes affecting the sciatic nerve are principally those of pressure neuritis, and the intervertebral foramen constitutes a critical region for symptoms arising from this cause. Chronic sciatic pain is usually caused by pressure or irritation of the sciatic nerve from an external source. According to Lewin,[4] predisposing factors are congenital anomalies, circulatory or endocrine disturbances, and trauma.

In the three cases reported here,* in which pressure on the sciatic nerve was caused by a fibrous cyst in the buttock, it would appear that the original cause was trauma, since all the patients had a history of injury preceding the

*Since this article was submitted for publication, I have found a fourth case which may be classified in this category. Mr. A. T. H., aged 57 years, complained of sciatica since 1929. During acute attacks he had difficulty when sitting. At the time of examination on April 27, 1948, the chief complaint was that he could not sit any length of time without pain and discomfort. Physical examination revealed an irregular mass between the anus and the tuberosity of the ischium, with continuity palpable to the greater sciatic foramen. Circum-injection with 1½ per cent Metycaine solution relieved the symptoms, therefore operation was deferred.

onset of symptoms. In two cases, the cystic mass was palpable, but in the other it was discovered by exploration, although suspected preoperatively, on the basis of the striking similarity of symptoms to those in the first two cases.

In the first two cases, the cystic mass was in the left buttock and in the third, on the right. In none of these instances was the sciatic nerve itself involved in the lesion. The situations of the lesions varied (Fig. 3), but in all they were close to the sciatic nerve so that, with certain movements, the nerve was subjected to abnormal pressure, producing severe pain. In the first two cases, it seems evident that the pain experienced by the patients in the sitting position was caused by direct pressure on the sciatic nerve. In the third case, the cyst was elongated, and the large quantity of fluid it contained shifted with motion. It is presumed that in this instance the pressure of this shifting fluid content produced the pain.

SUMMARY AND CONCLUSIONS

Three cases of sciatica, apparently initiated by trauma, are reported.

Surgical exploration in each of these cases disclosed a hematoma with cyst formation.

All three patients were relieved of the severe, radiating, sciatic pain by excision of the cysts.

These findings may be significant in certain cases of sciatica in which other causes of pain have been eliminated.

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AMPUTATION OF THE CANINE ATRIAL APPENDAGES

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THROMBUS formation in the left atrial appendage is a frequent sequela of rheumatic mitral disease,[1] and subsequent repeated arterial embolism often produces disabling or fatal complications. There are no effective methods to prevent such thrombus accretion or embolization therefrom. The use of the anticoagulant drugs, heparin and dicumarol, offers one possible solution of this problem. It has seemed to us, however, that in selected cases of repeated embolism a more direct approach might be feasible, namely, surgical excision of the thrombus-containing atrial appendage. As a preliminary step, the following experiments were performed to determine the effect of amputation of one or both atrial appendages of normal dogs.

METHODS

Experience and confidence in this type of cardiac surgery were obtained in a series of previous experiments where, through the same approach, broad flaps of myocardium were elevated on each side of the descending coronary artery branches and sutured back over the vessels. This was done in an attempt to produce heart failure in the dog by thinning the effective functioning myocardium.

In the excision of the atrial appendages eight dogs were used, each weighing between 8 and 11 kilograms. Food was withheld for twenty-four hours prior to surgery. Under intravenous pentobarbital sodium anesthesia (25 mg. per kilogram) the animal was placed in the right lateral position and a skin incision made in the fifth or sixth left intercostal space. The incision was three to four inches long, running parallel to the ribs and extending from the left parasternal line to the left anterior axillary line. The incision was carried down to the parietal pleura, and as soon as the pleural cavity was entered mechanical artificial respiration was instituted and maintained throughout the remainder of the operation until the pleural cavity was closed. In the first few animals, portions of the fifth or sixth left ribs were resected. Subsequently, it was found that adequate exposure could be obtained by retraction of the ribs without rib resection. Following entrance into the pleural cavity, the left lung was retracted and protected with a towel moistened with isotonic saline solution. The left atrial appendage was easily seen through the parietal pericardium. The latter was incised longitudinally parallel to the left phrenic nerve, over the base of the appendage. One to two cubic centimeters of 20 per cent solution of procaine were applied topically to the atrial appendage to avoid ectopic atrial rhythms.[2] In some cases, procaine was injected directly into the atrial myocardium in the region of subsequent amputation. An Allis clamp was then placed on the tip

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of the atrial appendage, and a right angle clamp or curved hemostat clamped across the base of the appendage. In two dogs, two transfixion sutures were placed below the clamp and tied securely. In the remainder of the dogs, simple ligature at the base of the appendage sufficed. The atrial appendage was then amputated with scissors, and a transfixion suture was placed as reinforcement distal to the encircling ligature. Atraumatic needles and 00 catgut or braided silk sutures were used. There was no significant bleeding. The parietal pericardium was incompletely closed with interrupted silk sutures, leaving a 2 cm. aperture in the base of the pericardial sac allowing drainage into the pleural cavity. The parietal pleura was closed, artificial respiration discontinued, and the chest wall repaired in the usual manner. After closure of the chest residual air was aspirated by needle and syringe. Intravenous papaverine (15 mg.) was used in some cases to stimulate spontaneous respiration when artificial respiration was discontinued.

Exactly the same operative procedure was followed when the right atrial appendage was excised, except that the animal was rotated to the left lateral position after the pericardial cavity had been entered. In five animals, the left atrial appendage, in two the right atrial appendage, and in one both atrial appendages were removed.

Postoperatively, the animals were placed in individual cages, and 200,000 units of penicillin were administered intramuscularly daily for ten days. Serial electrocardiograms (three standard limb leads, and precordial leads from the apex and the third right parasternal intercostal space, using the right foreleg or central terminal as the location for the indifferent electrode) were taken preoperatively, immediately after surgery, two weeks following surgery, and before sacrificing the animals, four to twelve weeks following the operation.

RESULTS

Seven of the eight dogs survived the operation. The first animal developed respiratory difficulty, and the heart ceased beating 15 minutes after chest closure. The other seven dogs had an uneventful postoperative course. Within twenty-four hours after surgery they were ambulatory and eating well, and within one week they were lively and behaved as did nonoperated dogs.

The serial electrocardiograms did not reveal any abnormal atrial rhythms. The P and T deflections varied somewhat in direction and amplitude from record to record. These variations are ascribable to changes in position of the animal and its heart,[3] although an attempt was made to keep the animal in the same position each time a record was taken. There were no abnormal deviations of the S-T-T or P-Ta segments which would indicate significant injury to the ventricular or atrial myocardium. One animal developed a broad, notched P wave in Lead II immediately following surgery, indicating intra-atrial block. This abnormality had disappeared by the time the subsequent electrocardiograms were made. The P-R interval remained unchanged in all animals.

Two dogs were sacrificed four weeks postoperatively, the remaining animals after twelve weeks. Necropsy revealed firm adhesions between the visceral and

parietal pericardium, only in the region of the stump of the atrial appendage. Elsewhere, the pericardium was smooth and glistening. In the dogs in which both atrial appendages were removed, there were thin, fibrous pleuropericardial adhesions in the region of the incision of the pericardial sac. The atrial incisions were firmly healed in all dogs, and the endocardial surface was well endothelialized. There were no dilatations in the region of the amputation stump or adherent mural thrombi (Fig. 1). Careful examination of all organs failed to reveal congestion, emboli, or infarction. Sections were made through the atrial amputation stump and stained with iron hematoxylin and eosin, trichrome, and the

A. *B.*

Fig. 1.—*A*, Photograph of epicardial surface of atrial amputation stump showing firm, localized, fibrous adhesion of parietal to visceral pericardium (at arrow). *B*, Endocardial surface of amputation stump, showing complete endothelialization, and absence of mural thrombosis (at arrow).

van Giesen stain for elastic tissue (Fig. 2). The following report* was made: There was extensive fibrosis along the line of suture with formation of cellular connective tissue irregularly disposed and the seat of superimposed exudation of cells—lymphocytes, polymorphonuclear leucocytes, and some plasma cells. This extended through the muscular coat and the endocardium and involved the endothelial lining; the latter showed no mural thrombosis, and was re-endothelialized. In the vicinity of the ligatures, there was fibroblastic change and formation of richly vascular granulation tissue showing dense cellular

*Report by Dr. Simon Koletzky of the Institute of Pathology, Western Reserve University.

infiltrate. There were occasional foreign body giant cells, and also a moderate number of monocytes containing iron pigment. There were no abscesses. In general, the degree of inflammation was not severe.

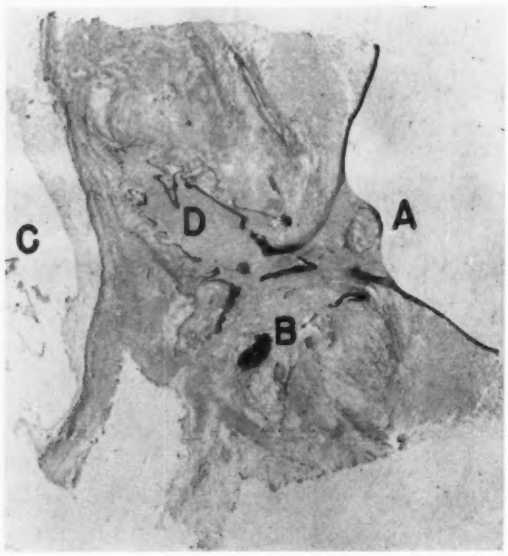

Fig. 2.—Van Gieson elastic stain of section through stump of atrial amputation, four weeks after operation (×5). *A*, Endocardial surface showing absence of mural thrombosis, with complete re-endothelialization; *B*, remnant of ligature; *C*, epicardial surface showing adhesion of visceral and parietal pericardium; *D*, extensive fibrosis along line of suture.

COMMENTS

The atrial appendages serve no significant function and their removal should not compromise the cardiovascular system. Amputation of one or both atrial appendages was successfully performed without subsequent mural thrombosis, embolism, persistent disturbance of rhythm, or congestive heart failure. The atria healed readily to produce a firm, well-endothelialized scar, and the myocardium united in a firm manner, histologically similar to that of muscle wounds elsewhere in the body. Beck and others[2, 4] have repeatedly emphasized this property of primary healing of wounds of the heart and the tolerance of both human and canine hearts to trauma.

We recognize the fallacy of transposing unreservedly the results of surgery on the heart of the healthy dog to the diseased heart of man. Moreover, the healing capacity of the dog differs considerably from that of man. However, the feasibility of atrial appendectomy in dogs suggests that further consideration be given to this method as a form of therapy in patients with rheumatic heart disease and mural thrombi in the atrial appendages. The ease with which the atrial appendages may be manipulated or excised also suggests that the atrial approach be used in surgical procedures on the mitral valve rather than the direct ventricular approach as has been used principally in the past.[4]

SUMMARY AND CONCLUSIONS

1. Seven of eight dogs survived unilateral or bilateral atrial appendectomy. One dog died immediately after the completion of the operation. The postoperative course was essentially uneventful in all surviving dogs. Serial electrocardiograms failed to reveal abnormal atrial rhythms or evidence of atrial injury except in one dog who developed transient intra-atrial block. Necropsy revealed excellent healing with a firm scar which was well endothelialized without subjacent mural thrombosis. Localized pericardial and pleuropericardial adhesions were found in the region of the amputation stump.

2. Excision of one or both atrial appendages is feasible in dogs.

3. It is suggested that clinical applications of this method be given further consideration.

4. The left atrial appendage approach for surgery or dilatation of the mitral valve is suggested in preference to the ventricular approach.

We are indebted to Dr. L. N. Katz for his criticisms.

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THE TREATMENT OF MALIGNANT LIP FURUNCLES WITH EMETINE

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THE common type of *furuncle* is usually defined as a circumscribed perifollicular suppuration followed by massive necrosis of the central hair follicle together with its sebaceous glands. When a similar process befalls not a single follicle but a continuous group of them the term *carbuncle* is generally used. Such a differentiation is, as I have pointed out over a long period, far from being correct.[1] In the first place, many typical furuncles are so voluminous in densely haired parts, as on the head, that for the process to occur in a single follicle is not possible. In reality the characteristic feature of a carbuncle consists in the formation of miliary abscesses in its periphery; under continuous dissemination of these miniature foci the carbuncular structure enlarges. The question how these miliary abscesses develop themselves cannot yet be answered in a satisfactory manner. Nevertheless, it is important to know that their spreading is not limited to the skin and the subcutaneous tissues but they may also involve deeper layers. Therefore, in advanced cases of neck carbuncle these miliary abscesses can infiltrate the underlying muscles. As satellites of malignant furuncle of the lip they make their appearance in the submucosa and the mimetic muscles. Even in metastatic manifestations, which often follow this type of staphylococcic infection, the same groupwise formation of miliary abscesses can be encountered. The so-called metastatic carbuncle of the kidney is its classic representative. In any rate it is a question of mere convenience for this special type of progressive purulent infiltration in the proximity of a furuncular structure to be called "carbuncle" or "malignant furuncle." It is astonishing that this typical anatomic feature which I first described in 1921,[1] and which can easily be verified when a carbuncle or malignant furuncle is thoroughly opened in due time, is not even mentioned in the current textbooks. Only in late cases, when the miliary abscesses have "melted" into large purulent tracts, the primary characteristic structure finally becomes unrecognizable.

The tendency to septic generalization is especially high in malignant lip furuncles. The rapid involvement of the adjacent veins—in the first place the vena angularis, with the well-known fatal consequences for the sinus cavernosus and the leptomeninges—is mostly responsible for it. Nevertheless, the development of septicemia is not necessarily bound to the competition of thrombophlebitis. When the initial furuncle once has assumed a malignant character—recognizable by the onset of fever often accompanied by chills and a quickly progredient and considerable swelling of the lip, severe pain, rapid deterioration of the general conditions—the chances for recovery are generally poor.

In the past, operative treatment has generally been regarded as indispensable in severe cases. However, for more than thirty years, conservative

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measures have been considered nearly everywhere as more promising. A short historical exposure on this behalf is to be found in my paper of 1926[2]. Typical of this predominant conservative tendency might be considered the formulation of *Ch. Lenormant*[3]: "The confinement to conservative measures helps the organism to limit the infection." My own experiences led me to quite different conclusions. In my previously mentioned article I attempted to explain that nonoperative procedure can be useful only in rather benign cases, whereas in the presence of real malignant development a positive result, if obtainable at all, can be expected only from a large débridement of the infected area combined with section of the angular vein. This latter is done by a transverse incision made 1 to 2 cm. beneath the interior angle of the orbita, dividing the soft parts down to the bone. For many years I have employed this method with fairly good results. However, I have since found out that emetine can be a precious drug against various septic conditions and this led me to try it also against malignant furuncles. The results I obtained were so satisfactory, that in the last cases operative measures could be entirely omitted. Before entering on the details of these experiments some previous explanation of this new use of emetine will be necessary.[4]

Emetine, the well-known alkaloid of the ipecacuanha, has been used for surgical purposes as an often successful drug against tropical liver abscesses due to amebic infection. Furthermore, it has been used occasionally against lung abscesses, and if a positive result occurs this has often been regarded as sufficient proof for the amebic nature of the suppuration. The good results I obtained in Ankara in cases of liver abscesses, which had no relation to previous amebic dysentery, made me doubt the exclusive antiamebic efficiency of emetine, and further experiences gave clear evidence that this drug can be very useful in many nonspecific infections. Especially good results were obtained in severe cases of peritonitis of different origin (appendicitis or traumatic lesions of the bowel), in severe biliary infections, in general septicemia, and in gaseous gangrene. In many of these cases emetine acted as an important supplement to surgical procedure, but sometimes no other treatment was needed.

The theory of this "new" therapeutic capability of emetine has still to be developed. The nearly momentaneous effect which often can be seen under these conditions is its most striking feature and could point to the assumption of a high antitoxic efficiency, for it cannot be regarded as a mere bactericidic one, as will be explained later. This is supported by experiments made in vitro by P. Bernoulli. Again, in vivo, that is, on infected animals, this author entirely confirmed my clinical experiences.[5]

In eight cases of malignant furuncles of the face, to date, I have used emetine—partly alone, partly as a supplement to failing surgical procedure. To give an idea of its efficiency a short report of those cases is indispensable.

CASE REPORTS

CASE 1 (Prot. No. 9355).—Naciye, 30 years of age, entered the hospital Sept. 27, 1943, with a malignant furuncle of the upper lip which began four days previously. She had had

several chills and the temperature was 39° C. A large incision was made. In the afternoon the temperature rose, accompanied by a new chill, to 39.5° C.; the following morning the temperature was again 39.5° C. The considerable local swelling persisted and the general state became alarming. Emetine (0.05 Gm. per day) was given. As Fig. 1 shows, this was followed by a rapid fall of temperature, which became normal in the course of a few days. Still more striking was the change in general condition, which became very satisfactory almost immediately afterward. Rapid diminution of the local swelling and the other inflammatory signs followed. The patient was discharged from the hospital completely recovered after ten days.

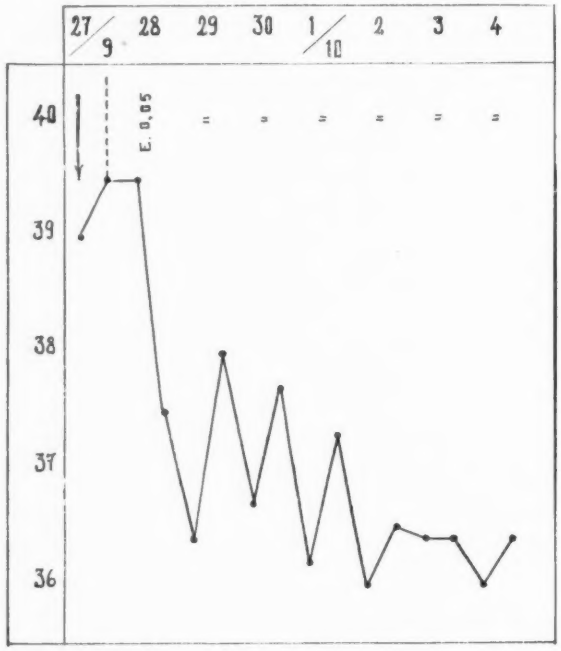

Fig. 1.

Case 2.—Orhan, 19 years of age, was admitted to another service in Ankara on Feb. 8, 1945. On February 3, a furuncle of the lower lip had made its appearance. The rapid increase of the local swelling together with repeated chills made stationary treatment necessary (February 8). Prontosil was given (Fig. 2), and on February 10 a large incision of the infiltrated area was made, but only a temporary diminution of the fever was obtained, whereas the local swelling showed a considerable increase. The general state became alarming. Albuminuria was 0.25 per cent. On February 13, the former incision was enlarged, the angular vein was cut (no visible thrombosis), and emetine (0.02 Gm. twice a day up to February 19) was administered. This was followed by a lytic decrease and final disappearance of the fever. Also, the local conditions and the general state showed a quick improvement. The patient was discharged home on March 3, as practically cured. In the urine there was still a trace of albumin. A short time afterward the recovery was complete.

Case 3 (Prot. No. 4121).—Izzet, 34 years of age, was admitted April 17, 1946, with a malignant furuncle of the upper lip. The alteration had developed in the course of three days. The lip showed an enormous snoutlike swelling, and the induration reached nearly the inner angle of the orbit. The general condition was serious. With superficial ether narcosis I opened the left side of the upper lip with a large frontal incision and it was found that all strata were crippled by miliary abscesses. Section of the angular vein was done

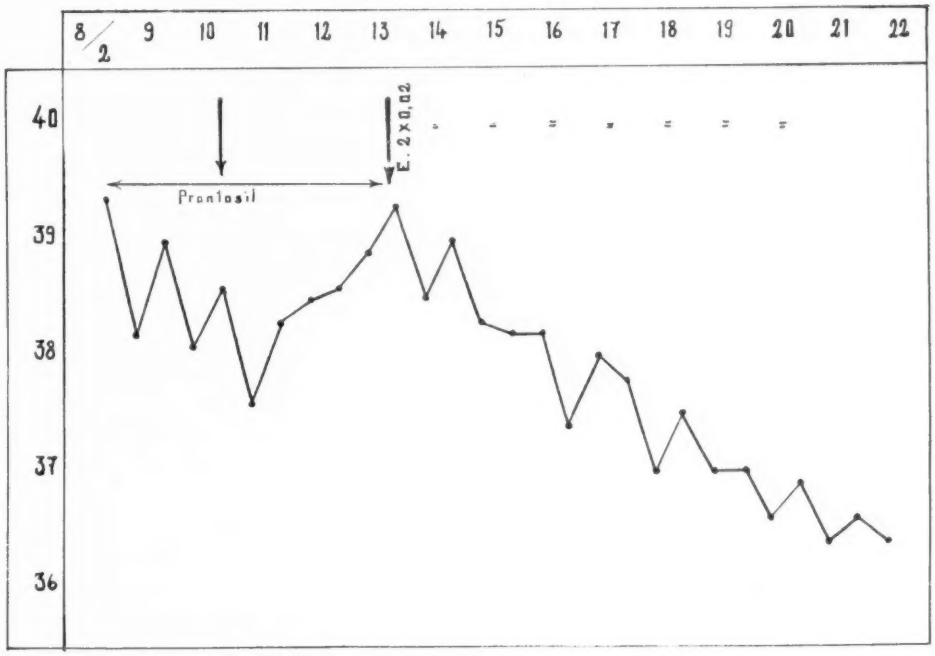
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Fig. 2.

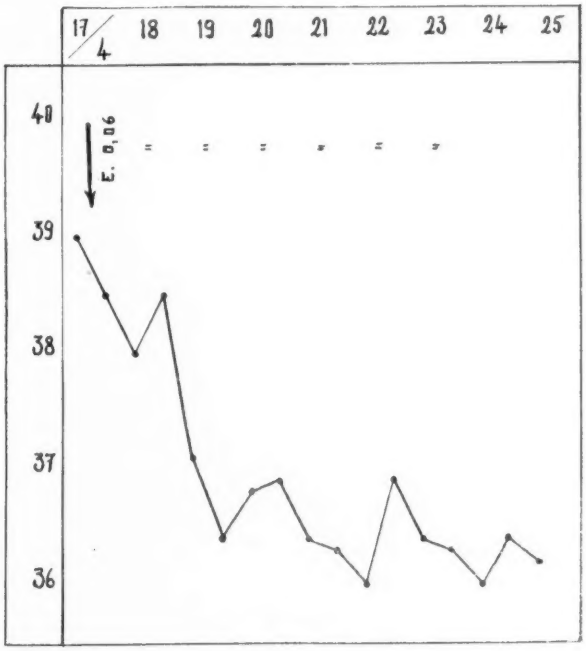
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Fig. 3.

and simultaneously emetine was given (0.06 Gm. per day) and continued up to April 23 (0.42 Gm. in all). The following day the patient felt well; the swelling was quickly decreasing. From April 20 he was definitely without fever (Fig. 3) and was dismissed as cured on April 25.

CASE 4 (Prot. No. 11955).—Semiha, 22 years of age, was admitted Oct. 21, 1946, with a malignant furuncle of the upper lip of five days' duration. There was pain, fever, and insomnia. At the time of admission, the whole lip showed a snoutlike swelling and the left cheek was also involved. Since the temperature was not very high (Fig. 4), with absence of chills, the treatment in the first days was only symptomatic. On October 23, the swelling showed a considerable increase and emetine was given (0.03 Gm. twice a day, continued till October 29, 0.42 Gm. total). As early as the following day the patient declared that for the first time she had slept well; the pain was entirely gone. Subsequently the infiltration diminished quickly and the general state was perfect. The temperature was normal from November 7. In the following days a small superficial abscess developed in the region of the cheek without any thermic reaction. On November 12 the focus was opened. The pus contained *Staphylococcus aureus*. In only three days the small wound was sealed. The patient was dismissed as cured November 14.

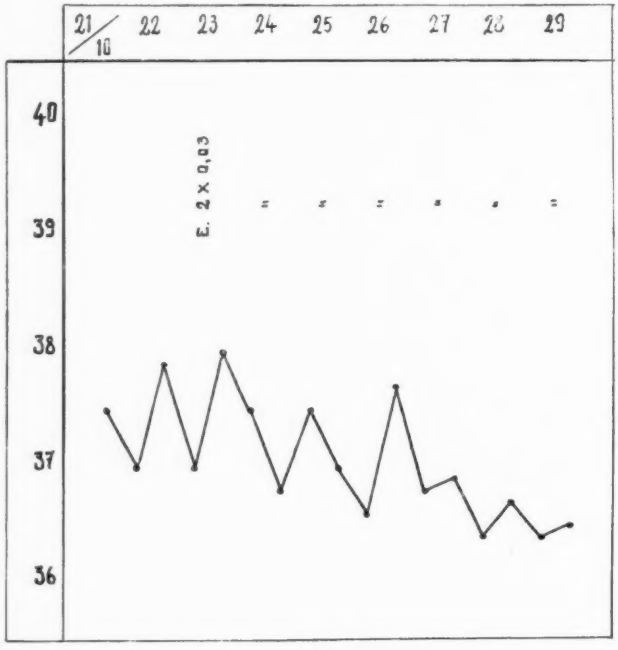

Fig. 4.

CASE 5 (Prot. No. 12989).—Izzet, 18 years of age, was admitted Oct. 9, 1947 with a furuncle of the upper lip of three days' duration. There was growing pain and swelling. Several times there was a feeling of shivering, but not a real chill. This was a typical malignant furuncle with large surrounding edematous swelling. The general state was not severely altered, and the temperature was only 37.4° C., with 17,200 leucocytes per cubic millimeter in the blood. Emetine (0.03 Gm. twice daily) was given. The following day the patient felt better. The second day the temperature was normal, and the swelling was diminished. On October 11 some pus was evacuated by spontaneous perforation of the skin. On October 13 the leucocytes were diminished to 9,400 per cubic millimeter. The patient was discharged from the hospital as cured on Oct. 15.

CASE 6 (Prot. No. 13098).—Hilmi, 32 years of age, was admitted to the hospital Oct. 11, 1947, with a furuncle of the lower lip. There was considerable edematous swelling, a general feeling of uneasiness and severe pain with insomnia. The temperature was 37.5° C. Emetine (0.03 Gm. twice daily) was given. This treatment was followed by immediate relief of pain and a quick diminution of the swelling. The patient was able to sleep without the aid of narcotics. The temperature became normal on October 14. The leucocytes were 11,600 per cubic millimeter on October 13 and 8,800 on October 18. The patient was dismissed as cured on October 19.

CASE 7 (Prot. No. 7357).—Firdevs, 16 years old, was admitted June 14, 1947. A furuncle of the upper lip had begun in a harmless way; about six days previously there was increase of the swelling and pain with strong headache, general bad feeling, and high fever. At the time of admission the temperature was 38.6° C. There was a snoutlike swelling of the upper lip with involvement of the cheek extending to within the region of the orbit. The general condition was poor. Immediate medication with emetine was instituted (0.06 Gm. the first day, 0.08 Gm. the following days, until June 21, a total of 0.62 Gm.). The changes which took place under this treatment were very striking. On the first day after emetine (Fig. 5) the temperature became practically normal. The patient declared spontaneously that a very short time after the first injection she had a wonderful feeling of well being. On June 17, the formerly large swelling was already reduced to the size of a small cherry; spontaneous perforation followed and the patient was discharged June 21 as cured.

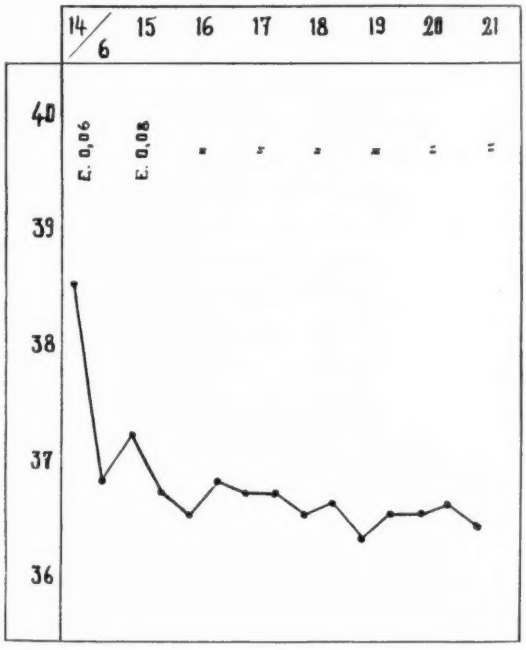

Fig. 5.

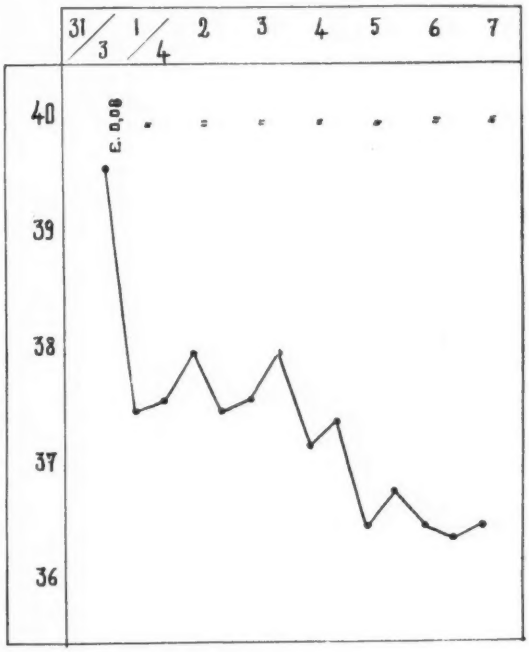

Fig. 6.

CASE 8 (Prot. No. 3854).—Sabit, 34 years of age, was admitted March 31, 1947, with a malignant furuncle of the upper lip. In the previous twenty-four hours there had been ten violent chills and when transferred on the ward he was under the grip of a long-lasting severe one. There was typical infiltration of the lip with widespread edematous swelling of the surrounding tissues. The temperature was 39.6° C. The urine contained a trace of albumin and some erythrocytes. The blood culture indicated the presence of hemolytic *Staph. aureus*. The patient arrived in the afternoon, 0.08 Gm. emetine was immediately given and the treatment was continued until April 7 (0.64 Gm., total). The following day

the temperature showed a critical decline to become normal four days afterward (Fig. 6). There was no recurrence of chills and all other signs disappeared in a short time. The patient was dismissed as cured on April 10.

These eight cases of malignant furuncle of the face represent the total of those patients treated with emetine, and in every one a very satisfactory result was obtained. In a critical analysis of them it should be considered that their clinical importance is not a homogeneous one. Therefore, in Cases 5 and 6, although belonging certainly to the class of malignant furuncles, the symptoms were mitigated. If the whole material had been of the same kind, it would be impossible to attribute the good final results with sufficient precision to the mere action of emetine. Similar doubts could be expressed for Case 3. Undoubtedly it was a very bad case, but a large incision had been made and no one could deny the possibility that the recovery might have been due to the surgical procedure. As I have explained before, I have been fervently partial to the surgical treatment of malignant furuncles, because the results obtained were very satisfactory. But the recovery which followed the incision in Case 3 was more prompt and in every way more impressive than ever before when incision alone was made. The assumption seems justified, therefore, that a considerable part of this favorable reaction must be attributed to emetine.

But even the most intransigent critic will have to acknowledge the really amazing effect of emetine obvious in the other cases. Very significant is Case 1. The initial incision was followed by a new rise in temperature, accompanied by a chill and growing local swelling. It may be that the incision had not been made to a sufficient extension. But nevertheless it is known, that under those conditions the prognosis becomes very bad. In this situation the administration of emetine was immediately followed by an impressive amelioration and quick recovery. This dramatic change means more than a mere coincidence. The same was seen in Case 2. Here, also, surgical intervention had been made before, but things took an alarming turn and the final recovery was certainly due more to emetine than to the completing of the primary incision. An equally rapid amelioration of highly critical conditions followed the administration of emetine in Cases 7 and 8. The most remarkable of them is undoubtedly Case 8. Not less than ten long-lasting chills had made their appearance in the last twenty-four hours, in this patient, and *Staph. aureus* was found in the blood. But this dangerous situation was entirely altered the following day. No chill recurred and final recovery took place in a short time. These last observations are so persuasive that no serious objection can be made against my interpretation. A further proof for this affirmation results from the fact that this reaction to emetine is not a phenomenon to be seen only in malignant furuncle. If emetine has any effect at all it very often assumes this dramatic form, may it be in cases of generalized peritonitis, severe biliary infection, purulent traumatic meningitis, or gaseous gangrene. In my former publications numerous examples of this kind of fulminant reaction are to be found.

It might also be mentioned that sometimes the patient spontaneously indicates the feeling of restored comfort even before the objective signs are manifested. In Cases 4, 5, 6, and 7 such a sensation was noticed.

It was already mentioned that a sufficient theoretical explanation on this behalf is still lacking. Nevertheless, the effect of emetine is not a strictly bactericidal one. In patients with widespread peritonitis a favorable reaction to emetine does not exclude the possibility that harmless residual abscesses might make their appearance afterward. As sequelae of septicemia those mitigated suppurations can, for instance, develop at places where injections, even of emetine, had been made. A characteristic example for this is seen in Case 4, where a small abscess still containing staphylococcus followed the acute furuncular infection.

The dosage of emetine hydrochloride is, for the adult, 0.03 Gm. twice daily up to 0.05 Gm. twice daily. Also when a full effect is obtained, the medication must be continued to the total amount of about 0.4 to 0.9 Gm. or even 1.0 Gm., for if interrupted too early recurrence of the infection can take place. When correct dosage is used, cardiovascular difficulties, as sometimes observed in dysentery cases, are not to be expected. In any case, the combination with a slightly stimulating drug might be recommended as a prophylactic measure.

CONCLUSION

Eight cases of malignant lip furuncles are reported. All of the patients recovered, mostly without any surgical interference, by mere use of emetine. The effect of the drug is generally a striking one. Even under the worst conditions, that is, when septicemia is already present, quick and complete recovery can be obtained. Emetine, which has given similar satisfactory results in numerous nonamebic septic infections of different types, can therefore be regarded as an important agent for the treatment of malignant lip furuncles.

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Erratum

In the article by Reynolds and Young, Jr., entitled "The Use of the Roux Y in Extending the Operability of Carcinoma of the Stomach and of the Lower End of the Esophagus" in the August, 1948, issue of the JOURNAL, the sentence beginning in the fourth line, page 260, "Whole blood administered at a rate of 1 liter an hour . . ." should read ". . . at a rate of ½ liter an hour . . ."

Case Reports

ENDOBRONCHIAL POLYPOID HAMARTOCHONDROMA

Review of the Literature and Report of a Case

R. W. Postlethwait, M.D., Winston-Salem, N. C., and R. F. Hagerty, M.D.,* and J. C. Trent, M.D.,* Durham, N. C.

ENDOBRONCHIAL polypoid hamartochondromas are rare lesions comprising only a small portion of all hamartomas of the lung. Only thirteen cases of this type have been reported. They arise as a polypoid growth within the bronchial tree and are composed principally of cartilage, with or without fat, epithelium, or connective tissue. The clinical and therapeutic differences between endobronchial and intrapulmonary hamartochondromas are considerable. An intrapulmonary lesion may remain asymptomatic for years, and grow to considerable size before pressure may cause symptoms. A small endobronchial lesion, however, may cause severe symptoms early, when of small size, because of the resulting obstruction. Also, an intrapulmonary lesion may require resection, whereas transbronchoscopic removal of the endobronchial tumor may result in cure. Because of these differences, the polypoid tumors are separated from the general group of hamartomas of the lung. Twelve cases of endobronchial polypoid hamartochondroma have been collected from the literature and are reported with one additional patient treated at Duke Hospital. The pertinent facts concerning these thirteen patients were abstracted and summarized in Table I.†

There are in the literature ninety-eight cases of hamartoma of the lung excluding the thirteen cases of the polypoid type. In 1926 Hickey and Simpson[2] collected thirty-six cases and reported two of their own. Verga[3] added twenty more cases in 1932 including three of his own. Twenty-three personal cases in addition to thirteen cases from the literature were reported by McDonald, Harrington, and Clagett[4] in 1945. Since that time Pouey,[5] Seifert,[6] and Simon and Ballon[7] have reported three cases. Schafer and Scott[8] recently reported an intrapulmonary hamartoma composed mainly of cartilage, removed by lobectomy. A solitary metastatic lesion from a bone tumor of the arm was suspected, but a benign lesion could not be excluded.

Pathology.—In 1904 Albrecht[9] stated "hamartomata are tumor-like malformations in which occur only an abnormal mixing of the normal components of the organ. The abnormality may take the form of a change in quantity,

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†Stordeur[1], in 1903, reported a hard tumor of the bronchial wall described as enchondroma. The information was inadequate to justify inclusion of this case.

arrangement or degree of differentiation, or may comprise all three. The deduction to be drawn from histologic examination of these formations is that they have originated in an abnormal mixing of the normal elements or from disturbance of their development.'' The exact origin of endobronchial polypoid hamartochondromas is in doubt. Eicken,[10] Blecher,[11] and Spiess[12] believed these tumors to be outgrowths of the bronchial rings. Siegert,[13] Spuler,[14] Paul,[15] and Moore[16] reported elastic fibers in the cartilaginous tissue of the tumors in their respective cases. Since elastic fibers are not present in normal bronchial cartilage, this was cited as evidence favoring an embryonal anlage as the site of origin.

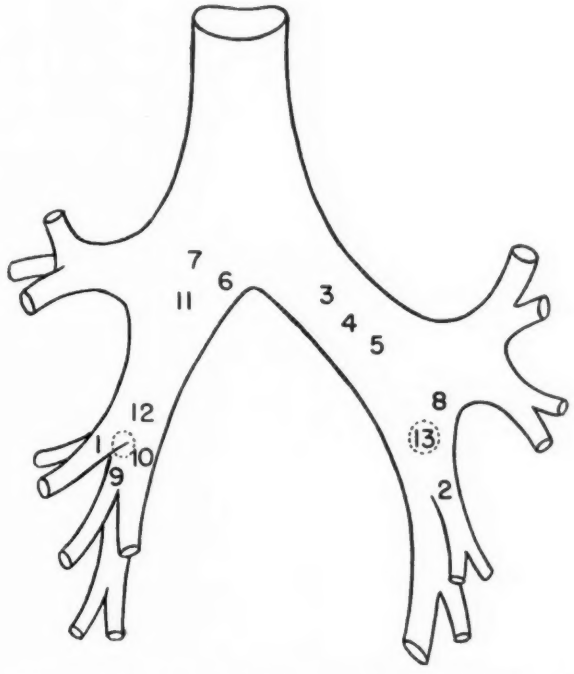

Fig. 1.—Showing location of thirteen reported hamartochondromas.

The location of the thirteen tumors is indicated in Fig. 1. Grossly the tumors appear bright pink in color, spherical in shape, with a nodular surface. The stalk may be more narrow than the tumor mass. The chondromas are firm and cut with resistance. Microscopically, abundant cartilaginous tissue is seen, frequently divided into plaques or islands by fatty tissue, cavernous blood spaces, and connective tissue. Ossification in some areas of the cartilage may occasionally be seen. The tumors are well encapsulated, and the bronchial surface covered by epithelium, usually cylindrical in type. There is no peribronchial erosion or invasion.

Clinical Manifestations.—In the series analyzed the average age of the patients was 54 years, the youngest was 21 and the remainder were over 40 years of age. Of the thirteen cases, eight were men, four women and the sex of one was not recorded.

TABLE I

AUTHOR	AGE (YR.)	SEX	SYMPTOMS	SIGNS	X-RAY	BRONCHOSCOPY	TREATMENT	PATHOLOGY
1. Siegert[13]	61	F	(Autopsy finding)					23 by 32 mm. in right middle lobe bronchus; cartilage surrounded by connective tissue, epithelial structures, and cavernous blood sinuses; abscess behind obstruction
2. Chiari[17]	68	F	(Autopsy finding)					20 by 20 mm. in left lower lobe bronchus, islands of calcification in hyaline cartilage, fat and scattered glands; bronchiectasis
3. Spuler[14]	?	?	(Autopsy finding)					40 mm. in left main bronchus; cartilage with elastic fibers, with dilated vascular bed, and connective tissue
4. Eicken[10]	41	F	Cough with purulent, blood-streaked sputum for 2½ yr.	Bronchiectasis with consolidation of left lower lobe		Visualized	Excision via bronchoscope; recovery	13 by 60 mm. in left stem bronchus; cartilage embedded in fatty tissue, covered with epithelium
5. Blecher[11]	21	M	Chest pain and cough with purulent sputum and fever for 3 mo.	Bronchiectasis on left	Opaque over left side		Open drainage of empyema; died	10 mm. in left main bronchus; ossified cartilaginous tissue; bronchiectasis and consolidation, left
6. Spiess[12]	47	F	Bronchiectasis with dyspnea and orthopnea for 4 yr.	Consolidation on right	Areas of increased density on right	Visualized	Excision via bronchoscope; recovery	40 by 10 mm. in right main bronchus; cartilage surrounded by connective tissue
7. Caussade, Surmont, and Lacapère[18]	53	M	Markedly productive cough for several months				Died	25 by 15 mm. in right main bronchus; cartilage; bronchiectasis

8. Paul[15]	69	M	Asthma since youth; abdominal fullness for 7 mo.	Fluid on left	Increased density on left		Thoracentesis; died	40 by 20 mm. in left stem bronchus; cartilage with myxomatous degeneration; bronchiectasis on left; portal vein thrombosis
9. Moore[16]	68	M	Urinary symptoms for 2 yr.; cough, wheeze and stridor for (?) 2 yr.	Râles and rhonchi bilaterally			Died	Tumor in right lower lobe bronchus, cartilage containing elastic fibers, embedded in fat; bronchiectasis; pneumonia both upper lobes and right lower lobe; carcinoma of prostate
10. Gebauer[19]	44	M	Cough and hemoptysis for 3 mo.	Râles in right lower lobe	Filling defect in right lower lobe bronchus (lipiodol)	Visualized	Excision via bronchoscope; recovery	8 mm. in right lower lobe bronchus; cartilage with plaques undergoing calcification
11. Davidson[20]	66	M	Cough, wheeze and dyspnea for 5 yr.; purulent sputum and hemoptysis	Consolidation of right lower lobe	Block of right lower lobe bronchus with terminal concave shape (lipiodol)	Visualized	Excision via bronchoscope; recovery	Tumor in right main bronchus; cartilage
12. Ulrich[21]	57	M	Recurrent pleurisy and bronchitis for 25 yr.; productive cough and weight loss	Râles and rhonchi in right lower lobe	Shadow in medial segment of right lower lobe	Visualized	Excision via bronchoscope; recovery	10 by 10 mm. in right main bronchus; fibromyxochondrolipoma; abscess distally
13. Postlethwait, Hagerty, and Trent	57	M	Cough and weakness for 3 yr.	None	Increased density superior segment, left lower lobe	Visualized	Lobectomy; recovery	10 by 10 mm. in superior bronchus of left lower lobe; cartilage; abscess distal to tumor

The duration of symptoms varied from three months to twenty-five years. The symptoms were grouped as those due to the tumor itself and those which were the result of bronchial obstruction, either partial or complete. The symptoms due to the tumor itself were cough, frequently severe, wheezing, chest pain and discomfort, dyspnea, sputum, hemoptysis or "streaking," and frequent chest colds. When the tumor occluded a large bronchus the distal bronchi were dilated and the lung behind the obstruction became atelectatic and infected. Pneumonitis, bronchiectasis, or lung abscess followed. The symptoms resulting from these changes were increased cough productive of purulent sputum, pleuritic pain, and dyspnea. The usual symptoms of toxicity as weakness, chills, spiking fever, and increased fatigability then developed. The final symptoms were those associated with an overwhelming pulmonary infection.

The local signs of the tumor partially occluding a bronchus were few. A wheeze which could be localized was occasionally present and aided in differentiation from bronchial asthma. Signs of recurrent attacks of pneumonitis were found. The signs associated with occlusion of a large bronchus were those of atelectasis, bronchiectasis, lung abscess, or empyema.

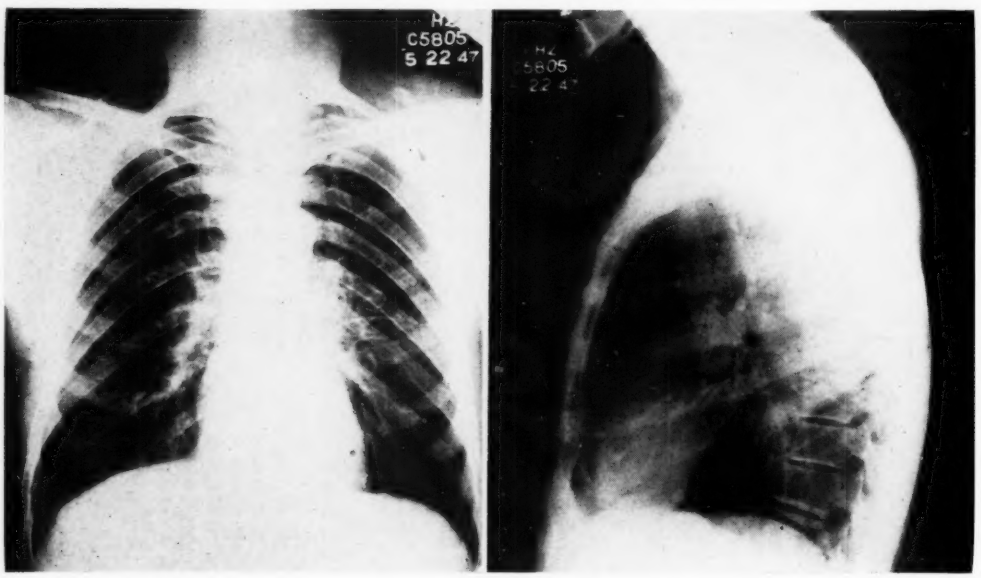

Fig. 2.—Roentgenograms of patient J. J. showing increased density due to abscess in superior segment of left lower lobe.

Diagnosis.—In the diagnosis of endobronchial polypoid hamartochondroma of the bronchus, x-ray examination was of value in revealing the changes associated with occlusion of a bronchus. Bronchial lipiodol studies showed failure of a lobe (Cases 7 and 10) or a segment (Case 13) to fill with the radiopaque material. In one patient (Case 11) the terminal concave configuration of the intrabronchial lipiodol suggested a bronchial polyp. The tumor was visualized in six instances at bronchoscopy (Cases 4, 6, 10, 11, 12, and 13).

Treatment.—Bronchoscopic removal was successful in five patients (Cases 4, 6, 10, 11, and 12). Lobectomy was performed in one (Case 13) because of inaccessibility of the tumor by bronchoscopy. The results in both types of treatment have been excellent. The remaining cases either progressed to a fatal issue or the tumor was an incidental finding at autopsy.

CASE REPORT

J. J., a 57-year-old white man, was admitted on May 20, 1947, because of productive cough and weakness of three years' duration. In 1944, he had a febrile illness diagnosed as "flu." From that time, recurrent episodes of fever with increased cough and weakness had occurred. For several months he had coughed up daily one-half cupful of odorless purulent sputum without blood. The physical examination was essentially normal. There was no anemia or leucocytosis. Sputum showed many polymorphonuclear leucocytes and many staphylococci and streptococci, which on culture were both hemolytic and nonhemolytic. The x-ray views are shown in Fig. 2.

Bronchoscopy showed a smooth, slightly lobular, pinkish, polypoid lesion arising from the posterior wall of the left lower lobe bronchus. Several attempts to biopsy the lesion were futile because of its location. Thoracotomy was performed under endotracheal ethylene anesthesia. The superior and medial segments of the left lower lobe were indurated. Left lower lobectomy was carried out. The postoperative course was essentially uneventful.

Pathology.—Grossly, a 10 by 10 mm., hard, pedunculated tumor arose from the most proximal part of the bronchus to the superior segment of the left lower lobe, producing obstruction to the bronchus and an abscess distally. The tumor was well encapsulated. Microscopic study showed a typical chondroma.

SUMMARY

Endobronchial polypoid hamartochondroma is a rare lesion, only thirteen cases having been reported. These tumors are clearly related to, but should be differentiated from, other hamartomas of the lung. The endobronchial polypoid hamartochondroma produces symptoms by partial or complete bronchial obstruction. If accessible, bronchoscopic removal is indicated. If inaccessible at bronchoscopy or if permanent pulmonary damage has followed the obstruction, lobectomy may be indicated.

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PRIMARY SARCOMA OF THE GREATER OMENTUM

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PRIMARY tumors of the greater omentum are infrequent, and primary sarcomas are much more so. Up until 1934 only seventy-five cases of primary sarcomas of the greater omentum were recorded; these were reviewed by Ranson and Samson.[1] Since that time other cases have been added by Sanes and Kenny[2] in 1934, Menne and Birge[3] in 1936, Levy and Pund[4] in 1940, and Lawler, Fox, and Cohen[5] in 1945. In view of the rarity of this neoplasm and the peculiar diagnostic difficulties associated with it, the reporting of another instance of the disease is appropriate.

CASE REPORT

F. K., aged 51 years, was admitted to the Wisconsin General Hospital, Aug. 9, 1946, with the chief complaint of pain in the abdomen. He stated that he had had a swollen abdomen for three to four years without symptoms. Eight months prior to admission, after eating a heavy meal, he developed right-sided abdominal pain, which was of short duration and disappeared spontaneously. Six weeks before admission he again developed abdominal pain, which was persistent and exacerbated by eating. During this period he noticed that the abdomen increased in size and became tense. He was conscious of a mass in the right side. Physical examination revealed a tender, tremendously distended abdomen, with a fluid wave and no palpable masses.

By peritoneoscopy no fluid was found in the abdomen, but in the right upper quadrant there were many fibrinous adhesions about a lesion considered to be metastatic, although no primary site was identifiable. Complete x-ray studies of the abdomen indicated a soft tissue mass in the upper midabdomen and showed a stomach which was high but which emptied with normal rapidity. The small bowel was pushed to the left, and the colon was unobstructed, although the left colon was displaced laterally. In the x-ray view of the chest the diaphragm was abnormally elevated with some compression atelectasis in the bases of both lungs.

The laboratory findings included a normal urinalysis, hemoglobin 9.7 Gm. per cent, red blood cells 3.9 million, and white blood cells 18,100, with the following differential: 82 per cent neutrophiles, 12 per cent lymphocytes, and 6 per cent monocytes. Blood sugar, nonprotein nitrogen, and icterus index were within normal limits. Prothrombin was 100 per cent, and Hanger's test negative. Serum protein was 6.8 Gm. per 100 c.c. with an albumin-globulin ratio of 3.8/3.0.

A laparotomy, performed Sept. 3, 1946, disclosed a large fungating mass which obscured the viscera. There was considerable bleeding on exploration, so that the exact limits of the tumor could not be safely determined. A biopsy was taken, however, and subsequently reported as a fibrosarcoma. The immediate postoperative response was satisfactory. Subsequently the patient's condition deteriorated gradually until death occurred one month later on Oct. 1, 1946.

Autopsy Study.—At autopsy the abdomen was filled with a very friable, lobulated, pale yellow, and partly necrotic mass, which was identified as the considerably enlarged and thickened greater omentum that had been transformed into tumor tissue. It extended from xiphoid to symphysis and was bounded by the lateral parietes on either side. In areas it measured in thickness up to 14 cm., and it weighed 20 pounds. After removing all of the tumor mass, numerous small transplants, 2 to 4 cm. in diameter, were found scattered over the peritoneal surface. There was a single small metastasis measuring 5 cm. in the dome of the right lobe of the liver, and another on the pleural surface of the right diaphragm.

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Microscopic Examination.—Microscopically the neoplasm was composed preponderantly of large, spindle-shaped cells, compactly arranged in linear and whorled groups (Fig. 1) and intimately associated with numerous small capillary vessels, many of which contained erythrocytes. The cell borders were vague, and the cytoplasm was acidophile, opaque, and many contained clear, structureless vacuoles (Fig. 2). Mitotic figures were common. In areas, vacuoles of several cells approximated and appeared to coalesce. Reticulum was not conspicuous. The nuclei were large, spindle-shaped, hyperchromatic, and occasionally were indented and flattened by the vacuoles. This histologic pattern was replaced occasionally by a myxomatous type of tissue, in which the spindle cells were few, multiprocessed, and appeared to float in the homogeneous acidophile matrix (Fig. 3). The various metastases resembled the parent neoplasm closely except the one in the liver, which had a very vascular structure (Fig. 4). In its center the vessels were large, cavernous, and filled with erythrocytes. Peripherally the vessels were smaller, with walls composed of two or three cells, and empty lumina. On the edge adjacent to the liver tissue, the cells were vacuolated and similar to those seen in the omentum, although here the vascular arrangement was more evident.

Fig. 1. Fig. 2.

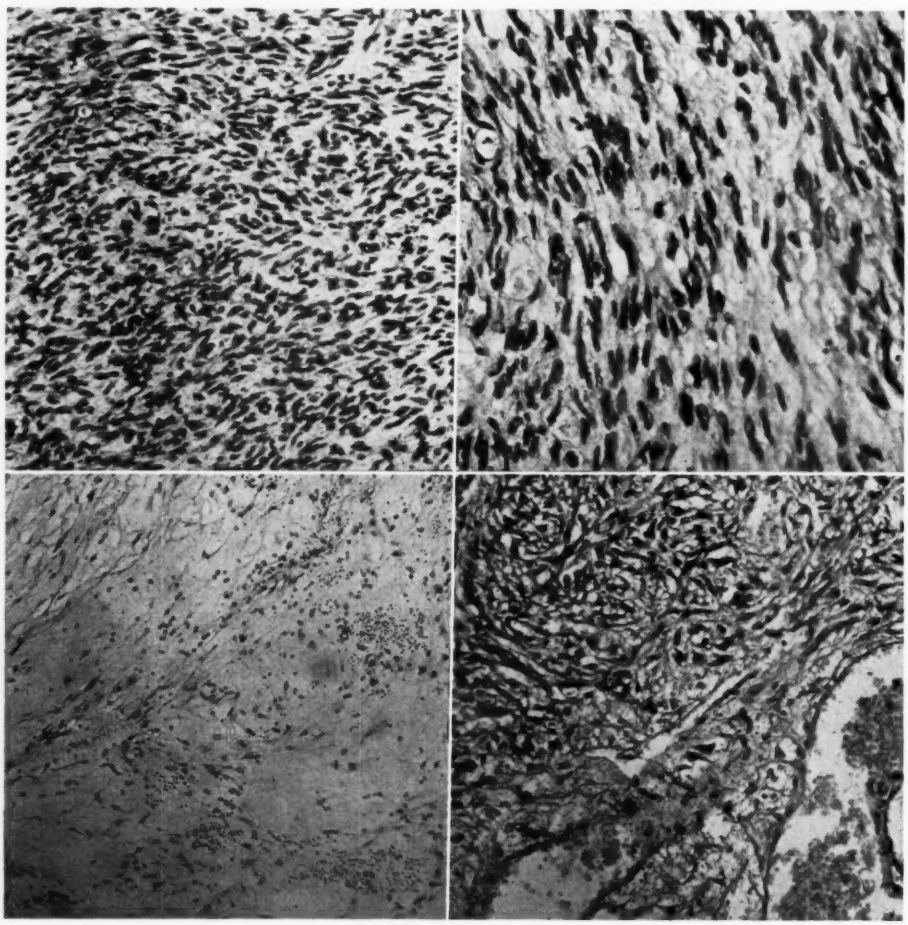

Fig. 3. Fig. 4.

Fig. 1.—Section from omental neoplasm, in which the spindle-shaped cells are conspicuous. (×150 hematoxylin and eosin.)

Fig. 2.—Section from omental neoplasm in which the vacuoles and their relation to capillaries are seen. (×300 hematoxylin and eosin.)

Fig. 3.—Section from omental neoplasm from a myxomatoid portion, with spiderlike cells and homogeneous matrix. (×150 hematoxylin and eosin.)

Fig. 4.—Section from metastatic nodule in the liver, at the junction of the cavernous-like central portion and peripheral capillary angiomatous tissue. (×300 hematoxylin and eosin.)

DISCUSSION AND CONCLUSION

This tumor falls grossly into the category of the diffuse type as indicated by Levy and Pund.[4] It satisfies the criteria proposed by McDonald[6] for authentication of omental sarcoma, in that its extent in the omentum indicates this structure as the origin, and that histologically it is a sarcoma, which has metastasized. The interpretation of the significance of the vascular pattern requires circumspection; it would be simpler certainly to follow McDonald's example and classify this as sarcoma merely, until the vexed question of the endotheliomas is settled. There are, however, several facts which deserve fuller consideration. Most significant are the formation of intracytoplasmic vacuoles and the fusion of such vacuoles from several cells to form a distinct cell-lined space. This pattern of cell behavior resembles that described by Kettle and Ross[7] in their cases of vascular endotheliomas. Of considerable importance in the interpretation of the tumor is the clear-cut vascular structure of the hepatic metastasis, which in some parts resembles a cavernous angioma. Whether this is regarded as a source or an extension of the neoplasm, its vascular nature confirms the origin of the tumor as endotheliomatous in this instance. It is pertinent to indicate that in none of its parts does the lesion resemble a liposarcoma, either histologically or cytologically (Stout[8]), and it is unlikely that fat stains are of use in excluding liposarcoma any more conclusively than cytologic criteria because, whereas liposarcomas may be poor in fat, degenerative sarcomas may contain much fatty material. It is, moreover, possible that infiltrative neoplasms incorporate adipose tissue, a contingency most likely to be encountered in predominantly adipose tissues such as the omentum. The tumor under consideration complies with the criteria of a primary omental sarcoma and conforms in its structure to the endotheliomas described by Kettle and Ross.[7] It closely resembles those designated as endotheliomas by Levy and Pund.[4] While liposarcoma cannot be rigidly excluded, it is unlikely because of the absence of certain cytologic features.

SUMMARY

A case of primary sarcoma of the omentum is described. It is classified as an angioendotheliosarcoma, because of its peculiar structure and the obviously vascular nature of the metastases.

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Editorial

Traumatic Dislocation of the Hip

WHILE it is not a common accident, traumatic dislocation of the hip is a potent source of permanent disability. The circulatory damage which produces the incapacity can occur after partial dislocation, fracture-dislocation, central dislocation, or the more frequent posterior and dorsal dislocations. From 30 to 50 per cent of those who receive this injury suffer late symptoms in the hip, such as pain, stiffness, and pronounced limp. The old statistics on the frequency of such dislocations are of no value now because of the constantly increasing number of automobile accidents which produce them.

Since the time of Hippocrates, surgical textbooks have described the anatomic types of displacement, the methods of diagnosis, and the various manipulations needed to reduce dislocations. Unfortunately, there was no mention of the more important aftercare or the need for protection of the joint during the late stages of healing. As a consequence, this injury was almost always followed by preventable destructive changes in the joint. Only in the last ten or twenty years has sufficient knowledge been gained about the circulation to the hip to indicate proper aftercare of these cases. Studies on the circulation of the hip after fractures of the neck of the femur have revealed the precarious blood supply of the head of the femur and its marked vulnerability to injury. This knowledge has demonstrated the need for long protection of hips that have been dislocated. Moreover, it has been observed that those patients who have received other injuries that required long bed rest have ultimately gained better hip function than the patients who bore weight early.

Roentgenograms of a dislocated hip, of course, do not reveal the extensive soft tissue damage or the amount of displacement of the head of the femur at the moment of the accident. Modern anesthetics, supplemented with curare and other relaxing drugs, make it possible to reduce hip dislocations without undue force. In fact, gentle manipulations are adequate in most cases and they decrease the danger of further damage to the soft tissues. The significance of this is that there is seldom any need for open reductions of dislocations of the hip. Even when there are fractures through the head of the femur, it is generally possible to replace the hip without open operation. The initial maceration of the tissues about the hip, the extensive hematoma, and the subsequent open reduction are frequently followed by pathologic calcifications about the joint, with ultimate stiffness.

After reduction of dislocations of the hip, the patients should be kept in bed for two or three weeks or until the soft tissues have recovered from the effects of the accident. Then crutches or a weight-bearing brace should be used for many months. If there has been an accompanying fracture of the acetabulum, it may be necessary to maintain traction on the leg or to place the

patient in a body cast for six or eight weeks. The main point of treatment, however, is that the patient should not bear weight on the involved extremity for at least six months from the time of the accident. While this is a very difficult rule to enforce, patients can be convinced that unless this course is followed they are likely to have serious permanent disability. The progress of healing of the bone can be followed by frequent roentgenograms to observe the density or contour of the head of the femur. Even after a period of six months' protection and after the patient is allowed to begin weight-bearing, he should have roentgenograms taken every two or three months for one year to make certain that no late destructive changes are developing in the head of the femur. If degenerative changes begin to appear, it is necessary for the patient to resume the use of crutches.

The recognition of the great importance of circulatory changes following traumatic dislocations of the hip has completely revolutionized the plan of treatment. It has focused attention on the total inadequacy of former treatment and the possibility of reducing the residual permanent disability that follows this serious injury.

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San Antonio, Texas

Recent Advances in Surgery

CONDUCTED BY ALFRED BLALOCK, M.D.

AN ANALYSIS OF THE TREATMENT OF TROPHIC ULCERS

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SINCE the time of Hunter, treatment of trophic ulcers has always been a matter of speculation, and, as one reviews the literature, realization of this fact is borne out, for there is a manifold amount of prescribed, as well as the so-called "specific," treatment for similar as well as identical ulcerative lesions. However, it is not within the scope of this paper to catologue the entire gamut of trophic ulcers, but only to bring to the fore one type with which the general practitioner, as well as the surgeon, will have to contend, as an aftermath of World War II. These are the trophic ulcers which arise secondary to peripheral nerve injuries. In our series, the majority of the latter represented a complete anatomic severance of the nerve, with but few instances of physiologic dehiscence only.

Our purpose, then, is to summarize a routine of treatment which we have found to be most efficacious, after having observed and treated some 650 patients, who exhibited a total of 733 nerve lesions.

The types of patients treated were essentially all battle casualties, with nerve injuries to the extremities; the age group varied between 18 and 30 years, and each had been in continual hospitalization for some two to three years following injury.

It is agreed that the presence of certain skin changes in these patients associated with long-standing nerve injuries, and in themselves loosely designated as "trophic" in nature, are very definitely and unequivocally the result of disuse of the extremity, rather than of definite vascular damage and impairment. This was further borne out by an analysis of these cases, which demonstrated that, although most all of them were accompanied by, or occurred concomitant with, fractures, an associated vascular injury was the exception rather than the rule. Certainly, less than .1 per cent of the patients required a major vessel ligation at the time of injury. This, then, allows us to suppose that these were all true trophic ulcers, in accordance with the strict definition of the term. In addition, wherever sympathectomy was carried out, it was so done because of an associated reflex sympathetic dystrophy, rather than being executed specifically for purposes of increasing the peripheral circulation.

The opinions or assertions contained herein are the private ones of the authors, and are not to be construed as official or reflecting the views of the Army Medical Department or the Army at large.

*Formerly Captain, Medical Corps, Army of the United States.

The ultimate pursuit of our discussion will, therefore, include anatomic considerations, etiologic factors, and treatment thereof, since our primary premise was the diminution or absence of painful and thermal stimuli to the involved areas. The presence of hypesthesia or varying degrees of anesthesia, whatever was the prevailing situation, made for the numerous injuries (especially burns) sustained by these patients to the already affected member.

In our anatomic considerations, we are primarily concerned with the two components of the sciatic nerve, namely, the posterior tibial and common peroneal nerves; and the three main peripheral nerves of the upper extremity, that is, the median, ulnar, and radial components of the brachial plexus. According to this anatomic division, many patients will then be seen to have sustained multiple injuries, some to a single extremity, others to any combination of the four.

LOWER EXTREMITY

In so far as the lower extremity was concerned, paralysis of the posterior tibial nerve (Fig. 1, *A*) rendered itself most problematic, especially regarding the prophylaxis of ulcer formation. This we found of markedly more significance than any and all treatment which we could offer, once a disease process had been established. A good common sense regime of preventative treatment appeared, then, to be the sine qua non, and all measures instituted were directed along these channels. Personal neglect and ignorance of the seriousness of the situation on the part of some of the patients were also obstacles which we had to overcome.

As one can readily see, the sensory distribution of the posterior tibial component of the sciatic nerve is in an area which bears the entire weight of the body, and is most vulnerable to trauma, extreme thermal states, and pressure either from ill-fitted shoes, or full weight-bearing over bony prominences. In several cases we had also to contend with residuals from trench foot, with some patients who had active infections of epidermophytosis, and with still others who had a disturbed circulation secondary to severe cicatrices.

Our greatest problem was the development of callosities over bony prominences, especially over the heads of the metatarsals. If these callosities were not closely observed and early treatment instituted, they usually followed a progressive course, with ultimate undermining and subsequent ulceration and secondary infection. Prophylactic treatment then being of primary concern, the following regime was outlined and to this we rigidly adhered.

Mental awareness was first and foremost in our teachings to the patient. He was constantly reminded of the fact that he had to watch closely and protect the involved extremity, for there were no longer the so-called "messengers" in his foot to warn him of the onset of any deleterious or menacing stimuli.

Foot hygiene was another "must" on the agenda. The feet were to be bathed twice daily, followed by a thorough dusting with talcum or regulation foot powder. White socks were preferred and were to be changed daily.

When partial or full weight-bearing was subsequently allowed, the problem of shoes was very much individualized and required special construction for

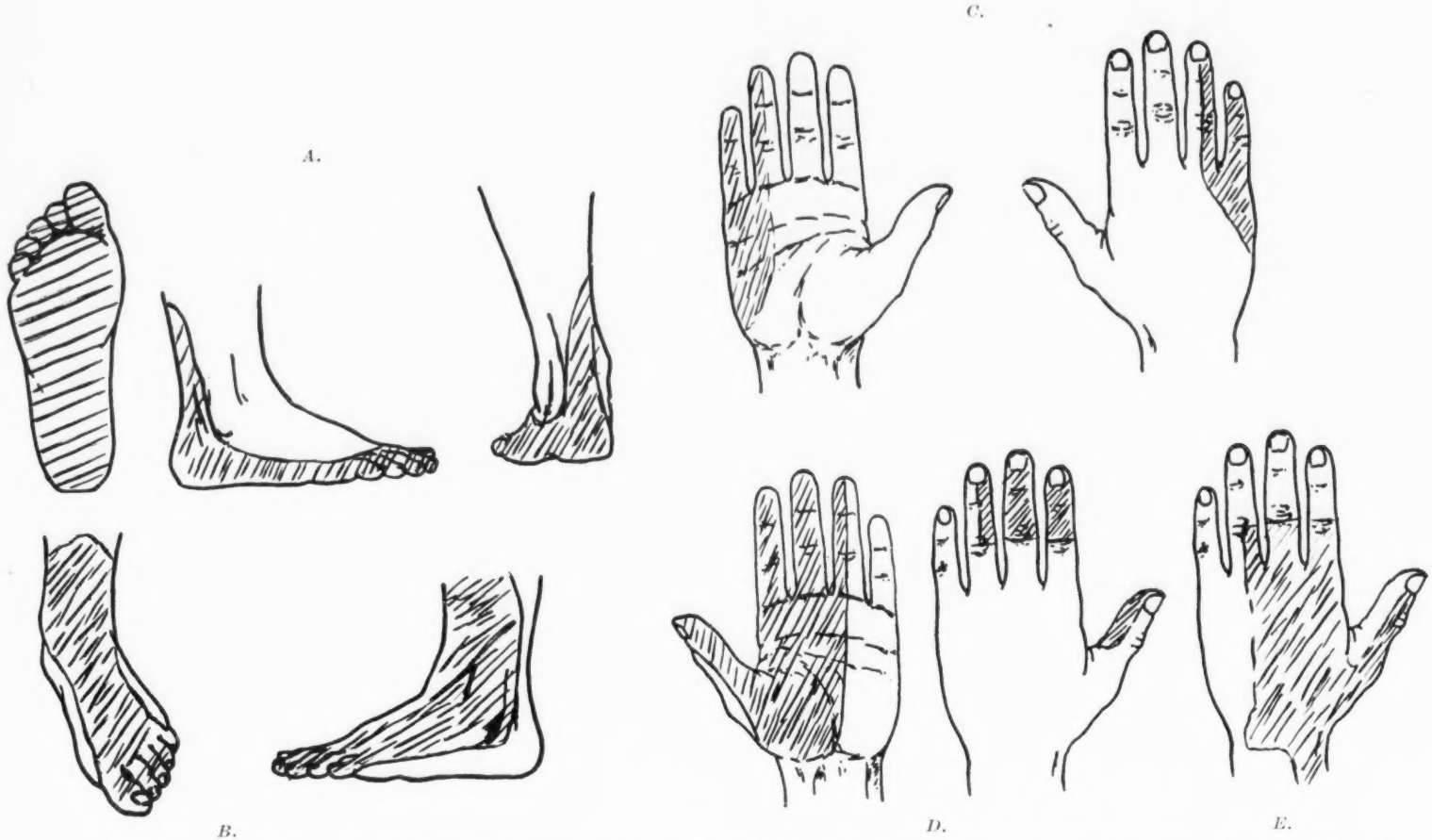


Fig. 1.—*A*, Sensory loss resulting from posterior tibial nerve paralysis. *B*, Sensory loss resulting from common peroneal nerve paralysis. *C*, Sensory loss resulting from ulnar nerve paralysis. *D*, Sensory loss resulting from median nerve paralysis. *E*, Sensory loss resulting from radial nerve paralysis.

each patient. Fundamentally, there were a few basic forms from which we worked, and from these developed a shoe which we felt satisfactory to conform to the special needs of the patient. These precautions were necessary in order to obviate any tightness, with consequent pressure by the shoe, for most of the patients developed a dependent edema after several hours of weight-bearing.

Fig. 2, *A* to *E*, serves to exemplify these particular walking aids. Because of the primary nerve paralysis, each shoe had also a brace attached. The further addition of a T-strap (Fig. 2, *F*) was necessary in those cases where there was varus or valgus deformity of the ankle.

The patient then either was prescribed an orthopedic convalescent shoe (Fig. 2, *A*) or else had the top of the low-quartered shoe split to the toe and a lacer adjustment applied (Fig. 2, *B*). When ulcerations developed over the insertion of the tendo achillis, the counter of the shoe was removed and a lacer adjustment applied (Fig. 2, *C*). Occasionally, when marked contraction of a single digit occurred, as usually happened to the great toe, we had but to excise a section from the dorsum of a normal shoe to prevent pressure in this region (Fig. 2, *D*).

In cases with plantar callosities or healed ulcerated areas, sponge rubber or felt insoles were inserted, cut out and tapered to conform with the callus or bony prominence on the plantar aspect of the foot (Fig. 2, *A* and *E*). It was further noted that shoes had to fit snugly, but not tightly, in order to obviate friction burns which usually resulted from a loosely fitted shoe.

Patients were divided into four categories, according to the type of active therapy required, as follows:

Category I: Cases with hyperkeratotic callosities but with no evidence of infection.

Category II: Ulcerative lesions with secondary infection.

Category III: Ulcerative lesions with secondary cellulitis and/or lymphangitis.

Category IV: Ulcerative lesions secondary to burns.

Before considering each individually, however, the following routine requirements were insisted upon in all categories:

1. In any case, weight-bearing on the involved extremity was not permitted.

2. During the course of dressings, adhesive tape was never to be applied directly to the skin on the involved extremity. Such a procedure, in the presence of certain trophic changes, might add an additional insulting stimulus.

3. No shoe was to be worn on the involved extremity.*

4. (a) Elevation of the extremity to a level of 20 to 30 degrees above the horizontal was to be carried out for at least six to eight hours during each waking day.

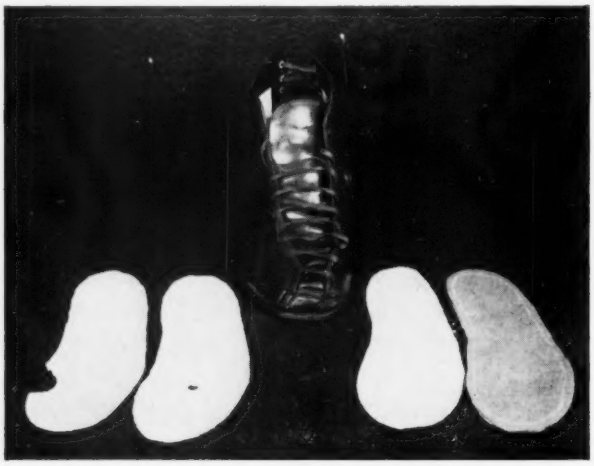
(b) Ambulatory patients were never permitted to place the extremity in a dependent position over prolonged periods of time. When sitting, the foot and leg were to be elevated on a second chair or stool, to at least the horizontal position.

5. Fifteen to thirty minutes of active exercising of the involved extremity,

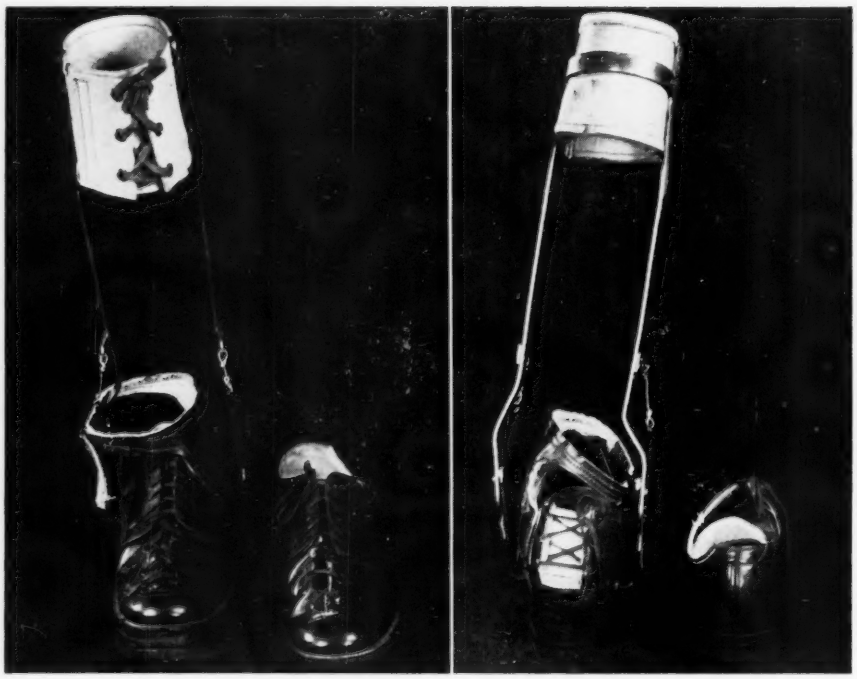
*Many patients privately secured a fur-lined, soft leather or felt "boot," which they wore when up and about, to protect against the minimal amount of everyday trauma.

four times a day, was mandatory. This was carried out, as much as possible, under the direction and guidance of the physiotherapy department.

Those patients considered under Category I, as outlined, were then treated according to the following regime:



A.

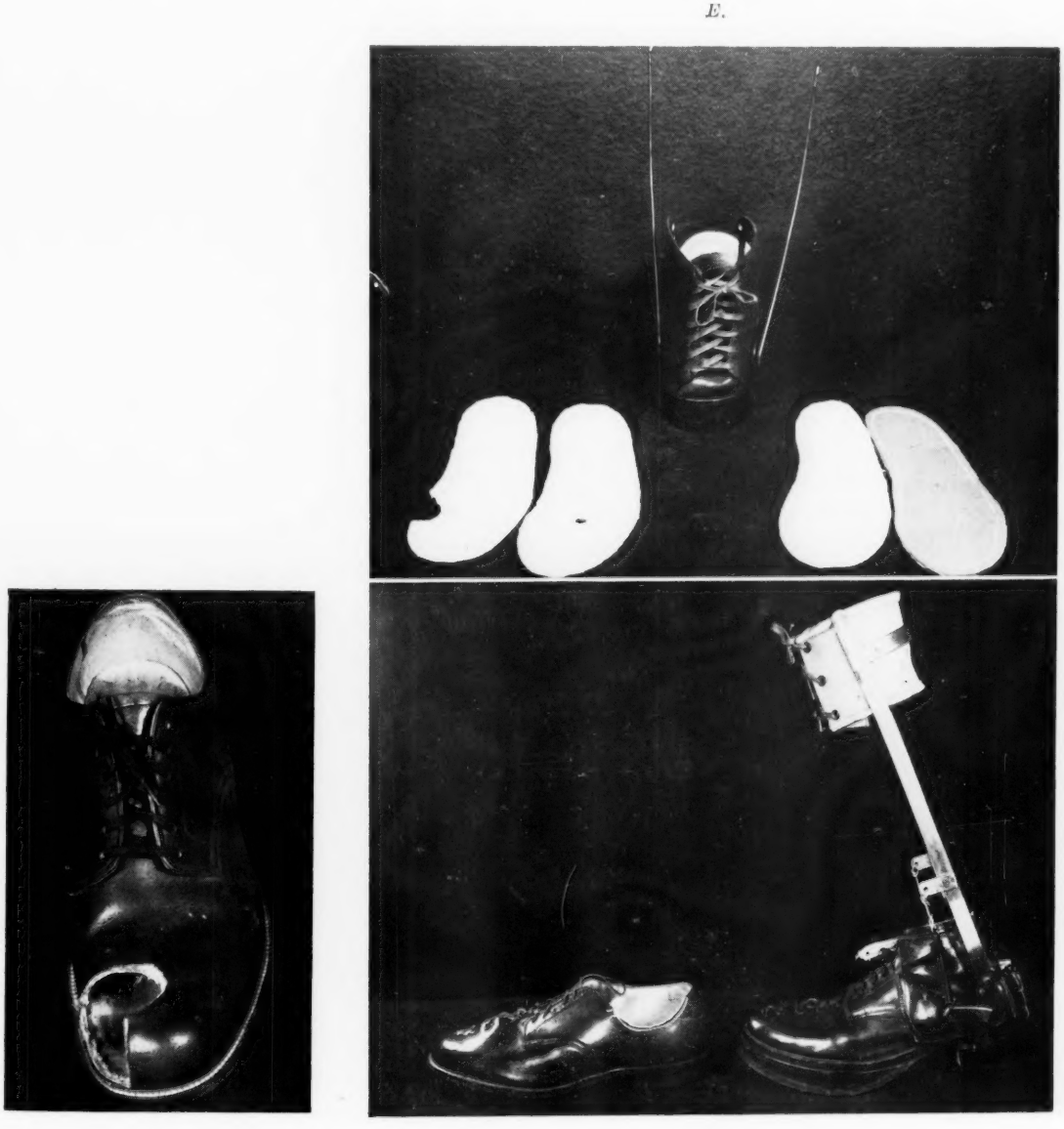


B. *C.*

Fig. 2, *A*, *B*, and *C*.—*A*, Orthopedic convalescent shoe (anterior view) and specific felt insoles. *B*, Low-quartered shoe, split to toe and lacer adjustment applied. *C*, Low-quartered shoe, counter removed and lacer adjustment applied.

Warm (5 per cent) boric acid soaks were applied to the involved area, three times a day. A petrolatum gauze dressing was applied to the calloused area, after each soaking. When the callus had become well demarcated, it was trimmed to its base. In order to return "tone" to the soft tissues, the foot was then given alcohol massages three times a day, for one week.

Following this, the patient was fitted with a suitable shoe, such as has already been described, and was then ready to begin gradual weight-bearing.



E.

D.

F.

Fig. 2, *D*, *E*, and *F*.—*D*, Low-quartered shoe with dorsally excised wedge only. *E*, Orthopedic convalescent shoe (posterior view) and specific felt insoles. *F*, T-strap on lateral aspect of low-quartered shoe for varus deformity; brace and lacer adjustment previously applied.

This was, at first, limited to thirty minutes daily, but later increased by degrees until full weight-bearing on the involved extremity was finally tolerated.

Lesions assigned to Category II required the selection of those therapeutic agents which demonstrated an antiseptic, as well as a keratinogenic, effect. Those considered most efficacious were iodoform gauze, scarlet red ointment, and chloresium, and were employed in this order. Accordingly, we found that in but a very few instances did we have to change our therapeutic agent. This exhibited itself mainly when the patients manifested a definite idiosyncrasy to the drug used, or in those instances where they were resistant to it.

The exact mode of treatment consisted of the following: The weeping wound was soaked in (5 per cent) boric acid solution for two hours daily, after which one of the afore-mentioned therapeutic agents was applied locally. This procedure was then continued until the ulcer bed showed evidence of healthy granulation tissue, with progressive epithelization, and a relative drying of the wound. When this had manifested itself, soaking was discontinued and only the particular therapeutic agent applied. In most instances, this was iodoform gauze. The latter was then continued until the ulcer had become fully epithelialized. Final treatment consisted of proper shoe fitting and progressive weight-bearing, as previously outlined.

It is best to interject at this time that, in several cases, there was noted a severe local reaction, ascribed to the use of iodoform gauze. This manifested itself by swelling, redness, vesiculation, and pruritus. These manifestations were, however, well controlled by the use of Burow's solution (1:1000) soaks and the oral administration of calcium gluconate tablets.

Category III, namely, those ulcers complicated by a cellulitis and/or diffuse lymphangitis of the entire extremity, presented a somewhat more comprehensive problem.

The patient was kept at absolute bed rest, with temperature, pulse, and respiration recordings every four hours. A white blood count and sedimentation rate were taken twice weekly. The involved extremity was elevated on two to three pillows, and was kept in continuous hot packs for approximately forty-eight hours. Application of a penicillin pack (500 units per cubic centimter) directly to the ulcer site preceded the usual boric acid solution wet dressing. This period of time was found usually sufficient to abort the acute phase of the infection, without unduly macerating the adjacent soft tissues. Since this usually involved the treatment of many almost completely anesthetic areas, it was absolutely imperative that the temperature of the solutions used be rigidly regulated so as to obviate the additional insult of superimposed burns.

In addition to the local application, penicillin was also administered subcutaneously, usually in doses of 100,000 units every three hours, and for such period of time as was necessary to reduce the white blood count and sedimentation rate to normal levels. At this time there was, most usually, a complete remission of the cellulitis and/or lymphangitis present.

After cessation of the acute phase, the patient was continued on the regime exactly as outlined under Category II.

Ulcerative lesions occurring secondary to burns, namely, those considered in Category IV, focused our attention primarily on the resultant ulcer. Since the involved area rarely exceeded 5 cm. in diameter, shock per se was never of therapeutic consequence.

Our course of treatment for these lesions was one of débridement, with the subsequent application of a softening agent, usually petrolatum gauze. Dressings were changed on the average of two to three times a week, since more frequent disturbance of the wound would tend to retard, rather than stimulate, healing. Scarlet red ointment, applied to those ulcerative areas where delayed healing was a factor, appeared to enhance the healing processes.

COMMON PERONEAL COMPONENT

In so far as the common peroneal component of the sciatic nerve was concerned those ulcers which occurred were usually in its autonomous domain (Fig. 1, *B*). These resulted primarily through neglect and came about as an aftermath of overzealous or faulty treatment, usually at the hands of the patient himself. Many times, he might thoughtlessly employ the use of a hot water bottle, or place the injured extremity against a hot radiator or heater, being grossly unaware of the resulting burn to the anesthetic area. Callosities, arising secondary to pressure from ill-fitted shoes, were also a cause of subsequent ulceration in this group.

The treatment employed for both burns and secondary ulcerations was exactly as that already described.

UPPER EXTREMITY

In considering the upper extremity, we found that burns were of primary concern, and that lacerations and other traumatic lesions were secondary. Any or all of these were usually brought about following an attempt to use the injured extremity, for one or another reason. Cigarette burns were perhaps most common. Where there was ulnar nerve involvement (Fig. 1, *C*), the patients incurred the burns by having rested the anesthetic hand on a hot object.

Perhaps the largest group of cases herein concerned were those with median nerve injury (Fig. 1, *D*). As previously stated, it was the burning cigarette which was most often the offending agent, with involvement of the index and long fingers. Radial nerve injuries per se seldom rendered themselves problematic and were of small concern in our series (Fig. 1, *E*).

Those ulcerations of the upper extremity which were resultant from burns, as well as any other superimposed complications, were treated exactly as outlined previously for similar conditions of the feet.

DISCUSSION

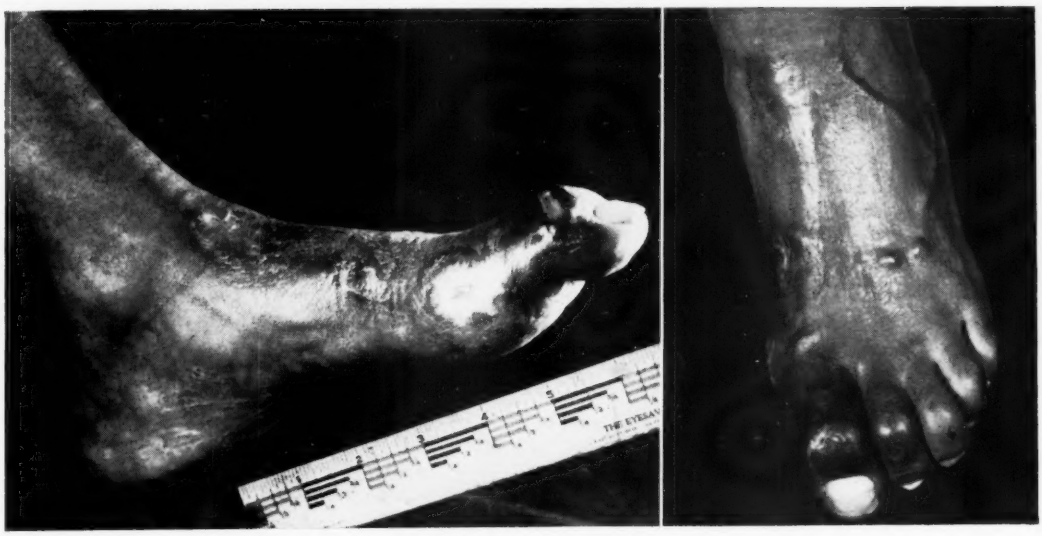
The average length of treatment for those in Category I was approximately four weeks; those in Category II, ten weeks; those in Category III, twelve weeks; and those in Category IV, fourteen to eighteen weeks.

In Table I we have represented a complete analysis of all the patients treated over a period of one year, from Sept. 1, 1946, to Aug. 31, 1947. During

these twelve months, amputation was carried out in but five instances, and in one of these only because of a severe tendo achillis shortening and marked deformity about the ankle, which could not be corrected by surgery, in the presence of the nerve paralysis. All patients discharged showed a complete healing of the ulcers, and each had been given a trial period of full weight-bearing for at least forty-five to sixty days in advance of leaving the hospital.

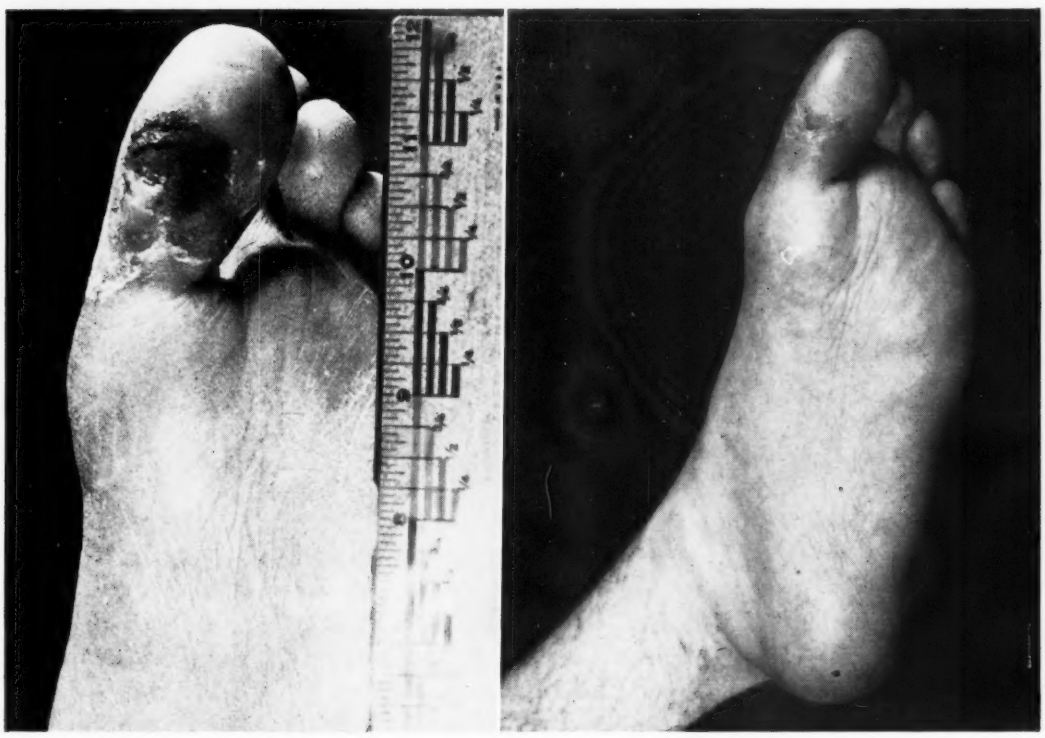
Fig. 3.—H. S. Complete paralysis of posterior tibial nerve (irreparable); marked excoriation and deep draining ulcer over heel; discharged on full weight bearing.

Figs. 3, 4, and 5 are representative of the type of lesion with which we were concerned. One of these (J. C.) required a below-knee amputation in spite of complete healing of a huge ulcer. This was necessitated because of the marked tendo achillis shortening and equinovarus deformity of the involved foot, together with an excessive degree of ankylosis about the ankle. Numerous attempts at correction of this deformity by conservative measures, that is, wedging in casts and splinting, were all unsuccessful, and, of course, surgical intervention was contraindicated because of the presence of an almost complete anesthesia. This unfortunate incident would then serve to stress a very



A. *B.*

Fig. 4.—R. N. F. (Colored). Complete sciatic nerve paralysis (repaired). *A*, Ulcerated and infected area on dorsum of great toe and medial aspect of foot, secondary to (third degree) hot water bottle burn. *B*, dorsal view showing areas completely healed; weight-bearing begun.



A. *B.*

Fig. 5.—J. C. Complete sciatic nerve paralysis (repaired). *A*, Huge ulcer, undermined and infected, secondary to wedging in cast following attempt to correct marked equinovarus deformity and ankylosis. *B*, Ulcer almost completely healed; below-knee amputation three months later because of unsuccessful attempts at correcting these deformities; weight-bearing impossible.

TABLE I

DESCRIPTION	NUMBER	PER CENT OF TOTAL
Total nerve injuries	733	
Number of cases	650	
Ulcers, upper extremity	7	0.95
Ulcers, lower extremity	70	9.5
Amputations	5	0.68
Sciatic nerve paralysis (complete)	171	23.3
Posterior tibial	47	6.4
Common peroneal	90	12.3
Brachial plexus paralysis (complete)	52	7.1
Radial	125	17.0
Median	126	17.0
Ulnar	122	16.6
Multiple injuries*	99	13.5

*More than one nerve in either extremity, *exclusive* of complete sciatic or brachial plexus paralysis.

cardinal point in the treatment of these patients, from the very outset of injury. That is to say, that conscientious and rigorous physiotherapy is indeed a very important procedure included in the regime of therapy. This should include both active and passive motion, within the limitations of the injury sustained, and, in addition, the very careful application of heat, together with massage and whirlpool baths, wherever feasible. Uniquivocally, these measures are of supreme importance if we are to adhere to the striking significance of a program of prophylaxis.

Examination of those extremities removed at surgery (amputation) very clearly demonstrated the pathology associated with nerve paralysis and the many secondary effects produced as a result of prolonged disuse of the involved member. In each, there was seen a focal fatty replacement of the muscle fibers with condensation of the nuclei into clumps and clusters, and in many areas a total absence of the fibers themselves. There was also a mild to moderate degree of inflammatory change with invasion by numerous round cells. The picture was truly one of tissue atrophy. In each, there was also an obliterative endarteritis of the smaller vessels, but nowhere was there vascular necrosis or significant alteration of the larger blood vessels. Pathologically, too, then, our original supposition that these were all ulcers of a true "trophic" nature seems further to be borne out.

In the final analysis, one can well realize that, associated with the healing processes of trophic ulcers secondary to peripheral nerve injuries, there must be the unrelentless courage of the patient, and the unwavering patience of the doctor. For, it is a matter of either saving the extremity or submitting the patient to amputation; and it may be said here, this would mean, in many cases, a patient with a bilateral amputation, because many whom we had been treating had a previous amputation of the member on the opposite side.

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Cardiovascular
Hypertension[1]
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Endocrine Disturbances
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Menopause—female, male

Nausea and Vomiting
Functional or organic disease (acute gastrointestinal and emotional)
X-ray sickness
Pregnancy
Motion sickness

Gastrointestinal Disorders
Cardiospasm[2]
Pylorospasm[2]
Spasm of biliary tract[2]
Spasm of colon[2]
Peptic ulcer[2]
Colitis[2]
Biliary dyskinesia

Allergic Disorders
Irritability
To combat stimulation of ephedrine alone, etc.[3,4]

Irritability Associated With Infections[4]

Restlessness and Irritability With Pain[3,4]

Central Nervous System
Paralysis agitans
Chorea
Hysteria
Delirium tremens
Mania

Anticonvulsant
Traumatic
Tetanus
Strychnine
Eclampsia
Status epilepticus
Anesthesia

HYPNOTIC
Induction of Sleep

OBSTETRICAL
Nausea and Vomiting
Eclampsia
Amnesia and Analgesia[6]

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Preoperative Sedation
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[4]Nembudeine®,
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[6]with scopolamine or other drugs.

SURGERY

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